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Original Contributions

Differential Diagnosis of Acute Poliomyelitis

A Four-Year Study of 717 Cases of Suspect Poliomyelitis

T. C. PAPERMASTER, M.D.
Minneapolis, Minnesota

THE Elizabeth Kenny Institute is a unique hospital in that it specializes in one disease.* The year 1952 brought the greatest deluge of patients to Elizabeth Kenny Institute in the history of the hospital. The clinical material was referred by practitioners throughout the Northwest area, including North Dakota, South Dakota, Minnesota, Wisconsin and Iowa. When the patient arrived at our Receiving Room, a history was taken, physical examination was performed by the medical staff, and a spinal puncture was done if it was deemed necessary, or if it had not been done by the referring doctor. If poliomyelitis could be ruled out at the initial contact, the family physician was called and the patient was sent either home or to another hospital with the approval of the family physician. By far, the greatest percentage of these people went home and were given follow-up care by the private doctor. A second group of patients, in whom the diagnosis was more obscure, was admitted to the Elizabeth Kenny Institute for further investigative procedures and, after that, if poliomyelitis could be excluded, the patient was sent elsewhere according to the wishes of the family doctor. The author became curious as to what happened to the individuals in whom that disease was ruled out. Letters were sent to referring physicians and to other hospitals requesting a final diagnosis on those patients. A similar technique was carried out for the years 1953, 1954 and 1955. This study presents a review of 717 patients who did not have poliomyelitis.

From the Medical Service, Elizabeth Kenny Institute, Minneapolis. E. J. Huenekens, M.D., Chief of Staff.

*The facilities of Elizabeth Kenny Institute were expanded in 1955 to include other neuromuscular disorders for rehabilitation. However, for the purposes of this paper the acute poliomyelitis suspects only were considered. The "acute" poliomyelitis patients we have arbitrarily defined as those patients seen on or before the 14th day of the onset of symptoms.

It was the belief of our staff that the listing of the disease entities in this series would make a "field study" of the differential diagnosis of poliomyelitis. This sort of thing had been done in the past,[1,2,3,4] and the only justification for this review is that it is a large series and it is of interest to the physicians practicing in this area who were kind enough to refer these patients. It will not be within the scope of this paper to discuss poliomyelitis itself, its symptomatology nor the literature.

No claim for absolute accuracy can be made, for the diagnoses in a large measure are dependent on the exactness of individual family physicians in diagnosing the patient at hand. The author, therefore, was forced to accept on its face value the report rendered to him by letter of the individual practitioner regardless of what his actual follow-up technique may have been. Those patients who were sent to other hospitals in the area for a more thorough investigation, particularly at the University Hospitals, Veterans Hospital and Minneapolis General Hospital, probably represent a more precise appraisal of the clinical state.

In addition, a few cases each year showed up in which the diagnosis was doubtful, and the patient was sent out only to have poliomyelitis confirmed elsewhere by further follow-up observations.

During the four-year period under consideration, 2,992 patients were sent to this hospital. Of these, 2,275 had acute poliomyelitis and 717 had some other disease, a ratio in the magnitude of three poliomyelitis to one non-poliomyelitis patient.

Table I represents the distribution according to age. It may be pointed out that 64 per cent of our poliomyelitis patients were in the age group below fifteen years and 61 per cent of our revised diagnoses of non-poliomyelitis patients were in the group below fifteen years.

Table II illustrates the distribution of cases according to the months of the year in which admission occurred. The interesting phenomenon here is that during many months of the year more

TABLE I. DISTRIBUTION BY AGE

	1952		1953		1954		1955		Totals	
	P	NP	P	NP	P	NP	P	NP	P	NP
Birth to 1 year	23	9	10	8	9	2	3	5	45	24
1 to 4	227	32	98	43	48	40	37	32	410	147
5 to 9	312	51	186	44	57	43	51	47	606	185
10 to 14	172	20	123	28	48	20	50	14	393	82
15 to 19	93	24	58	23	24	17	32	17	207	81
20 to 29	159	31	120	31	42	26	52	11	373	99
30 to 39	80	20	60	16	27	9	26	14	193	59
40 to 49	17	5	7	7	6	3	2	3	32	18
50 and over	8	3	4	8	0	3	4	2	16	16
Unknown	0	3	0	1	0	2	0	0	0	6
Totals	1091	198	666	209	261	165	257	145	2275	717

P—Poliomyelitis
NP—Non-Poliomyelitis

TABLE II. DISTRIBUTION BY MONTHS

	1952		1953		1954		1955		Totals	
	P	NP	P	NP	P	NP	P	NP	P	NP
January	16	4	5	4	2	3	3	3	26	14
February	4	6	3	1	0	4	1	1	8	12
March	2	5	1	2	0	8	3	7	6	22
April	0	3	1	2	2	2	0	11	3	18
May	0	8	2	7	0	13	3	11	5	39
June	6	5	10	6	1	14	3	14	20	39
July	79	17	84	33	28	32	20	24	211	106
August	355	64	243	72	85	33	119	40	802	209
September	332	23	198	44	91	32	58	19	679	118
October	197	32	67	26	37	15	36	8	337	81
November	75	25	43	7	8	7	8	5	134	44
December	25	6	9	5	7	2	3	2	44	15
Totals	1091	198	666	209	261	165	257	145	2275	717

P—Poliomyelitis
NP—Non-Poliomyelitis

TABLE III. DISTRIBUTION BY SEX

	1952		1953		1954		1955		Totals	
	P	NP	P	NP	P	NP	P	NP	P	NP
Male	618	90	370	96	144	89	152	78	1284	353
Female	473	108	296	113	117	76	105	67	991	364
Totals	1091	198	666	209	261	165	257	145	2275	717

P—Poliomyelitis
NP—Non-Poliomeylitis

non-poliomyelitis cases were seen than poliomyelitis cases. The high incidence of both groups occurring in July, August, September and October is parallel with the general experience with this disease.

Table III compares the sex ratio of our poliomyelitis patients and non-poliomyelitis patients. We had a distinct preponderance of male patients who had poliomyelitis. However, in the suspect cases, the ratio was approximately equal.

Categories of Disease

Table IV portrays a large group of patients in whom a wide variety of diseases and organ systems produced symptoms which led to referral to Elizabeth Kenny Institute. The largest percentage of this group are patients in whom there was failure to find any specific illness. A few in this so-called undetermined group were actually well children who were referred because of suggestive complaint associated with poliomyelitis exposure. Some were instances where no follow-up data could be obtained from the family physician and our own data were not conclusive enough. A small number of individuals in this group were thoroughly investigated and still an indeterminate conclusion was reached. It cannot be denied that some of these could have been abortive poliomyelitis.

Respiratory diseases (Table V) comprised about 37.7 per cent of the total group. A category which includes the following terms: influenza, "flu-like" illness, nonspecific upper respiratory infection, vi-

rus infection, and sinusitis were all lumped together. This is, admittedly, an inaccurate and obscure type of situation. However, these are the exact terms received from the physicians who resore throat which early may be indistinguishable from poliomyelitis. However, on the second, third and fourth day of the disease, the typical lesions with vesicles and ulcers appear on the anterior

TABLE IV. MISCELLANEOUS

	1952	1953	1954	1955	Total
Undetermined or no disease found	60	47	36	23	166
Acute rheumatic fever	6	2	3	2	13
Urinary tract infection	1	3	3	1	8
Gastroenteritis	3	5	3	5	16
Shigellosis	0	0	1	0	1
Typhoid	0	1	0	0	1
Infectious mononucleosis	2	3	2	0	7
Hepatitis	1	1	0	0	2
Pleurodynia	1	0	0	0	1
Periarteritis nodosa	1	0	0	0	1
Iliac adenitis	1	0	0	0	1
Sepsis	0	1	0	1	2
Scurvy	0	1	0	0	1
Myocardial infarction	0	1	0	0	1
Abcessed teeth	0	1	0	0	1
Furuncle on buttock	0	1	0	0	1
Allergic reaction	0	0	0	1	1
Mumps	0	0	0	1	1
Measles	1	0	0	0	1
German measles	1	2	0	0	3
Roseola	3	0	0	0	3
Totals	81	69	48	34	232

TABLE V. RESPIRATORY DISEASE

	1952	1953	1954	1955	Total
Influenza— "Flu"-like illness, non-specific upper respiratory infection, "virus", infection, sinusitis	41	64	21	14	140
Tonsillitis and pharyngitis	13	17	26	22	78
Cervical adenitis	4	0	1	0	5
Otitis media	4	2	2	2	10
Pneumonia	3	3	6	4	16
Bronchitis	0	3	0	0	3
Asthma	2	2	0	0	4
Laryngitis	2	1	0	0	3
Laryngotracheitis	0	0	0	1	1
Croup	1	0	0	0	1
Herpangina	0	4	1	3	8
Peritonsillar abscess	0	0	1	0	1
Totals	70	96	58	46	270

plied to our questionnaires. This can be appreciated only by one who practices general medicine or pediatrics and is well illustrated by a letter received from one of the prominent pediatricians of Minneapolis who stated concerning one of his patients that "Stephen . . . apparently had one of these crazy virus infections. He improved on antibiotics and is apparently entirely well."

The group labelled tonsillitis and pharyngitis was limited to those in which exudate and redness of the structures involved enabled one to make a more specific diagnosis. One syndrome occurring in this group forms a minimum number of cases statistically, although it would be found a great deal more if practitioners were aware of the clinical symptomatology. Herpangina caused by the Coxsackie virus is a July-August-September disease having abrupt onset of fever, headache and pillars and soft palate. When these lesions become manifest, the symptoms are usually gone and as a result the patient may not consult the physician when he is feeling better. Familiarity with this disease would minimize error in diagnosing poliomyelitis. However, we are also aware that Coxsackie disease and poliomyelitis may exist together in a patient, and each virus can be grown out of stool and throat washings.

The forty-five cases of orthopedic disabilities demonstrated in Table VI comprised about 6.5 per cent of our revised diagnoses, and most of these were brought in because of limb pain or back pain.

The psychiatric group in Table VII is the smallest one. However, an emotional component does occur in every individual with an illness. These patients are singled out because the

emotional part of the disease was the dominant one. All of these cases occurred in the second and third decade female, except for one ten-year-old male with an anxiety reaction and a fifteen-year-old male with petit mal.

TABLE VI. ORTHOPEDIC

	1952	1953	1954	1955	Total
Myositis (myalgia)	7	3	1	0	11
Myositis, of cervical erector spinae	0	0	2	0	2
Rheumatoid arthritis	4	0	0	0	4
Rheumatoid arthritis, hip	0	1	0	0	1
Ruptured intervertebral disc	2	0	1	1	4
Synovitis, leg	0	1	1	0	2
Back, sprain	2	0	0	0	2
Back, pain, cause not known	0	0	1	0	1
Torticollis	1	1	0	0	2
Legg-Perthes	1	1	0	0	2
Tendonitis of shoulder	0	1	0	0	1
Bursitis of shoulder	1	0	1	0	2
Knee injury	1	0	0	0	1
Leg trauma (hip)	0	0	2	0	2
Osteomyelitis, tibia	1	0	0	0	1
Osteomyelitis, calcaneous	0	1	0	0	1
Osteomyelitis, humerus	0	0	0	1	1
Ewing's sarcoma	0	1	0	0	1
Leg lymphedema	0	1	0	0	1
Muscle bruises	0	0	1	0	1
Epiphysitis, right capital femoral	0	0	0	1	1
Osteochondritis, left distal femoral epiphysis	0	0	0	1	1
Third degree burns of leg	0	0	0	1	1
Totals	20	11	10	5	46

TABLE VII. PSYCHIATRIC

	1952	1953	1954	1955	Total
Petit mal	1	0	0	0	1
Psychoneurosis	1	0	0	0	1
Hysteria	1	1	2	0	4
Malingering	0	1	0	0	1
Hysterical paralysis	0	0	0	2	2
Psychophysiologic disturbance with musculo-skeletal reaction	0	1	0	0	1
Conversion reaction with hyperventilation syndrome	0	1	1	0	2
Anxiety reaction	0	0	2	1	3
Totals	3	4	5	3	15

Of course, the most varied and interesting list of patients fall in the neurological disorders in Table VIII, and the author has subdivided them into infectious, neuropathies, vascular, traumatic, neoplastic and miscellaneous. Of the total, 21.5 per cent fall in this group. For some unknown reason, the years 1954 and 1955 brought the largest number of mumps meningo-encephalitis cases to confuse us. It was often necessary to await laboratory studies to give us the correct diagnosis in the case of lymphocytic choriomeningitis, western equine encephalitis, and mumps meningo-encephalitis (without parotitis).

One other interesting case deserves additional comment. A girl of eighteen years came in with central nervous system symptoms (headache, fever, stiff neck and back, sore throat) and a spinal fluid cell count of 8; protein 38 mg. per cent. Type 2 poliomyelitis was grown out of her stools, and she was discharged with a diagnosis of non-paralytic poliomyelitis after a fifteen-day hospital stay. However, three weeks later, the virus neutralizing antibodies against lymphocytic choriomeningitis showed a rise from 31 to 700. Therefore, in this peculiar instance, we were forced to make two diagnoses; this case is not included in our listing.

Comment

In reading over the panel of diagnoses imitating poliomyelitis, the reader may feel that it would be almost ludicrous to confuse certain of these entities with poliomyelitis. Indeed, careful history and physical examination could have avoided an unnecessary trip for some patients. However, an unreasonable suspicion of poliomyelitis occurred in a minimum of instances. Errors in over-diagnosing poliomyelitis are understandable with public pressure to contend with, and sometimes consultation with a center such as Kenny Institute becomes a "must" during the

season. An example of this is demonstrated by a letter from a general practitioner in a small Minnesota town on a 1952 non-polio patient who wrote, "I think we were a little too polio minded. We should have kept her home but . . . there was too much heat on."

We also had the experience of doing repeat spinal taps after the referring physicians had done a diagnostic puncture elsewhere, revealing elevated cell counts. To our surprise, many of our repeat counts have been in the normal range or much lower than that reported by the referring family doctor. Our interpretation to explain this discrepancy has been that the referring doctor or technician counted red cells and did not distinguish red from white in the chamber, or else the

TABLE VIII. NEUROLOGICAL DISORDERS

	1952	1953	1954	1955	Total
Neurological—Infectious					
Meningitis					
Aseptic with otitis and mastoiditis	0	0	0	1	1
Organism unknown	4	2	2	1	9
Pneumococcus	1 Type 1	0	0	1 Type 6	2
H. influenza	0	2	1	2	5
Meningococcus	0	3	6	1	10
Strep	0	0	0	1	1
Staph	0	0	0	1	1
Aseptic	0	0	0	5	5
Meningismus					
Cause not known	0	0	1	3	4
Post measles	0	0	0	1	1
Lymphocytic choriomeningitis	0	0	1	5	6
Meningo-encephalitis					
Cause not known	2	0	0	3	5
Mumps	2	1	15	18	36
Encephalopathy					
Secondary to U.R.I.	0	0	0	1	1
With pertussis	0	0	0	1	1
Encephalitis					
Measles	1	0	0	0	1
Chickenpox	0	1	0	0	1
Influenza	0	1	0	0	1
Unknown	0	10	2	2	14
Western equine	0	0	2	0	2
Totals	10	20	30	47	107
Neurological—Neuropathies					
Guillain-Barre	1	2	0	1	4
Transverse myelitis	1	1	0	1	3
Polyneuritis	0	1	2	0	3
Bell's palsy	1	0	1	0	2
Erb's palsy	1	0	0	0	1
Paralysis of arms, cause unknown	1	0	0	0	1
Radiculitis	0	0	0	1	1
Bulbar paralysis, cause unknown	0	0	0	1	1
Totals	5	4	3	4	16
Neurological—Vascular					
Hematomyelia	1	0	0	0	1
Cerebral vascular accident	1	1	0	1	3
Thrombosis of post inferior cerebellar artery	0	0	0	1	1
Totals	2	1	0	2	5
Neurological—Traumatic					
Head injury	1	0	0	0	1
Skull fracture	0	0	1	0	1
Totals	1	0	1	0	2
Neurological—Neoplastic					
Spinal cord tumor	4	0	2	0	6
Brain tumor	1	0	2	1	4
Totals	5	0	4	1	10
Neurological—Miscellaneous					
Convulsive disorder	0	1	1	0	2
Tetanus	1	1	1	0	3
Muscular dystrophy	0	1	0	0	1
Extradural abscess	0	0	1	0	1
Hemiparesis with U.R.I.	0	0	1	0	1
Headache, cause unknown	0	0	1	0	1
Herpes Zoster	0	0	0	2	2
CNS syphilis					
Meningovascular	0	1	0	0	1
Tabes dorsalis	0	1	0	0	1
Hydrocephalus with arachnoid adhesions	0	0	0	1	1
Totals	1	5	5	3	14
Neurological Total					154

cell count obtained off the chamber was multiplied by the wrong factor, consequently the exaggerated figure.

Summary

1. A review of 717 cases of suspect poliomyelitis, in which a revised diagnosis was made, is presented, covering a four-year period of admissions at Elizabeth Kenny Institute from 1952 through 1955. The disease categories are listed in tables.

2. Errors in diagnosing poliomyelitis may be minimized by the following methods:

(a) A careful history and physical examination.

(b) Familiarity with other disease entities which may be epidemic at the time that poliomyelitis is existing in the population. Examples of such diseases are: herpangina, roseola, and acute gastroenteritis.

(c) Care in the performance of a spinal puncture and in the examination of the resulting spinal fluid.

3. The important lesson to be learned here is that poliomyelitis in its early stages with borderline findings may be indistinguishable from many diseases in a vast spectrum. If, therefore, poliomyelitis can confound the experts often until the virus is grown out of stool cultures or until sufficient time has elapsed for serological data to be obtained, it is certainly reasonable to expect that many times the average medical practitioner who carries the greatest burden of patient loads may be at a loss, during the stress of the hour, to supply a diagnosis on a given patient. He may find it convenient and desirable to seek help from a center.

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N-ACETYL-P-AMINOPHENOL (TYLENOL ELIXIR) AS A PEDIATRIC ANTIPYRETIC-ANALGESIC

This study was concerned with pediatric clinical conditions in which an analgesic-antipyretic drug would be indicated, and evaluation of chronic toxic reaction.

Tylenol, an elixir containing 120 mg. N-acetyl-p-aminophenol per 5 cc., was administered to 121 infants and children with fever, pain, irritability and other symptoms of discomfort associated with a variety of upper respiratory and other infections (a total of twenty diagnoses.) In ninety-six of the patients antibiotics or sulfonamides were also used. Tylenol was administered for periods of one to nine days, the majority of the children requiring treatment for one to four days.

In this group Tylenol "produced effective antipyretic and analgesic responses and no evidence of side-effects that could be attributed to the medication."

An additional group of twenty hospitalized infants and children with tuberculosis were given Tylenol over a period of five weeks to determine its safety on prolonged use. In this group the total amount of the drug given to children under six years of age was 480 mg. (8 grains) per day; children over six received 960 mg. (16 grains) per day.

This prolonged administration produced no undesirable side-effects and had no effect on the hemoglobin concentration, total leukocyte count or differential leukocyte count. Special tests in five of these febrile patients indicated that prolonged administration of Tylenol had no effect on the prothrombin, bleeding or coagulation time.

In the entire series of 141 patients, Tylenol was considered "acceptable" or "liked" by 86 per cent of the children.—Cornely, D. A. and Ritter, J. A.: J.A.M.A., 160:1219 (April 7) 1956.

Function of the Endocrine Organs during the Postoperative Period

E. DAHL-IVERSEN
Copenhagen, Denmark

DURING the postoperative period practically all patients present a change in the normal endocrine balance. During recent years a group of my co-workers, E. Hasner, I. Engdahl, H. Paaby, B. Sorensen, T. Tobiassen, and I have studied this condition.

The changes occuring during the postoperative period are independent of the nature of the operation, as only very small interferences are possible without a change in homeostasis. Attempting to obtain as uniform conditions as possible, we have first and foremost used a series of cases subjected to gastric resection in our pathophysiological investigations. The clinical observations of the symptomatology of the postoperative period are based upon 871 patients subjected to widely different operations, with a postoperative course varying from the mildest to the severest degrees, which were fatal in some few cases. The pathophysiological investigations were made in 200 cases.

According to our investigations, sex and age have no influence on the metabolic changes during the postoperative period.

Changes caused by anesthesia are of quite short duration, and begin soon after anesthesia has been induced, having largely subsided when the actual, postoperative changes can be recorded.

The clinical picture developing after an operation will generally be manifest from two to six hours after the commencement of the operation. We considered that it would be expedient to divide the postoperative clinical picture into three groups: mild, medium severe, and severe cases.

Table I shows the criteria on which we have based this classification. Many objections may be made against such a classification, but it is based upon a comparison between the clinical pictures and the recorded pathophysiological reactions in the great number of cases I mentioned.

TABLE I.

Mild cases:
Rise of temperature to 38.5° for up to 5 days.
Increase of pulse-rate to 100 for up to 3 days.
Output of urine exceeding 350 ml. after 24 hours.
Respiratory rate not exceeding 20.
Intestinal passage suspended for 24 hours.
Blood pressure over 100 mm. Hg systolic.
Medium severe cases:
Rise of temperature to 39.5° for up to 5 days.
Increase of pulse-rate to 120 for up to 3 days.
Output of urine exceeding 150 ml. after 24 hours.
Respiratory rate not exceeding 25.
Intestinal passage suspended for up to 5 days.
Blood pressure over 100 mm. Hg systolic.
Severe cases:
All cases not belonging in the two preceding groups.

Considering the number of symptoms from the gastrointestinal tract, the circulation, respiration, urinary tract, from the nervous system, the psyche, the skin, the mucous membranes and the eyes, and considering the temperature which is observed during the postoperative period, the postoperative clinical picture in severe protracted cases presents numerous features in common with the pathophysiological responses in adrenal cortex insufficiency, whereas pictures resembling that of pituitary insufficiency develop only in the rarest cases.

The postoperative adrenal cortex function was investigated by systematic registration of the quantitative and qualitative changes in the excretion in the urine of 17-ketosteroids, corticoids and dehydroisoandrosterone, and by eosinophil counts in the blood.

The experimental period began three days before the operation and was generally concluded eight days after this. In complicated cases it was prolonged.

With regard to 17-ketosteroids, the preoperative average levels were somewhat lower than the average values stated by Hamburger, though within normal limits (Fig. 1).

Presented before the Saint Paul Surgical Society, Saint Paul, Minnesota, November 9, 1955.

Aided by grants from "Statens almindelige Videnskabsfond," "Kong Christian den X Fond" and "Nordisk Insulinfond."

This also applies to the findings in the case of the corticoids. The explanation is, inter alia, that most of our patients suffered from diseases of

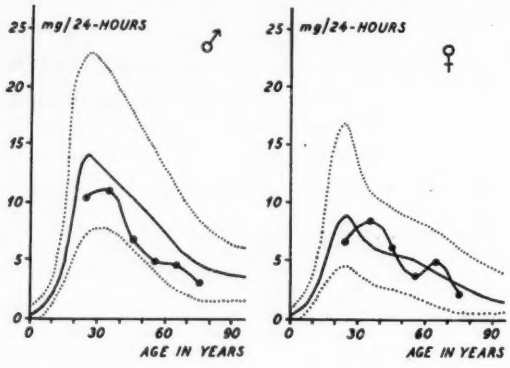


Fig. 1.

not occur until about the second or third day. The excretion falls to normal again between the third and ninth days, most frequently from the fifth to sixth day (Figs. 2 and 3).

In a number of cases the postoperative initial rise is followed by a fall below the preoperative level.

The one-third of patients who showed no significant rise in the 17-ketosteroid excretion all had postoperative eosinopenia and a significantly increased corticoid and dehydroisoandrosterone excretion. On the whole it appeared that the 17-ketosteroid excretion is the least sensitive indicator of an increased adrenal cortex function.

The corticoids of the urine originate chiefly or exclusively from the adrenal cortex, in contrast with the 17-ketosteroids which are also produced in the testes. Two biologically active corticoids, 17-hydroxycorticosterone and cortisone, have been isolated from the urine.

Within the first twenty-four hours after surgical interference or some other form of stress the

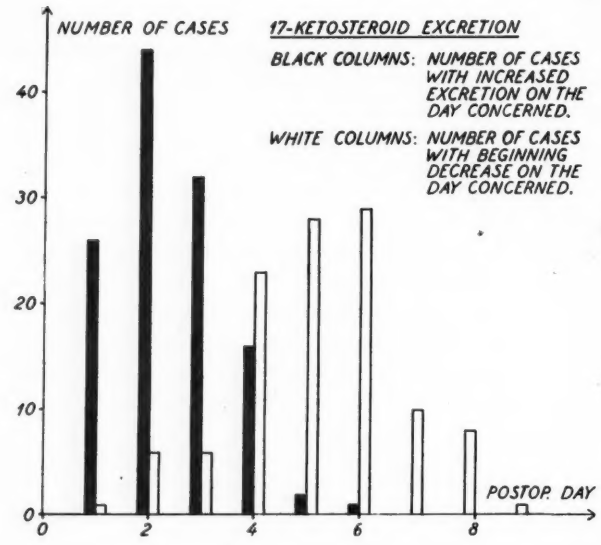

NUMBER OF CASES
17-KETOSTEROID EXCRETION
BLACK COLUMNS: NUMBER OF CASES WITH INCREASED EXCRETION ON THE DAY CONCERNED.
WHITE COLUMNS: NUMBER OF CASES WITH BEGINNING DECREASE ON THE DAY CONCERNED.
POSTOP. DAY

Fig. 2.

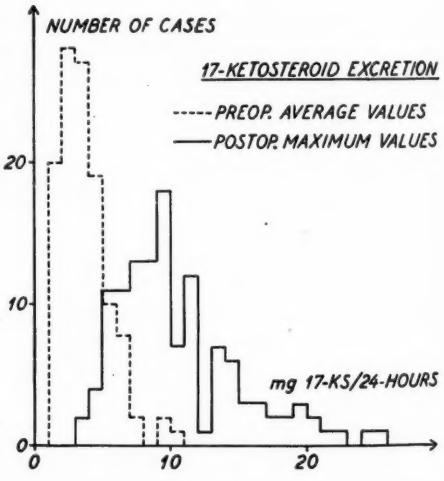


Fig. 3.

chronic nature, and according to investigations of recent years such patients have a lower excretion of these substances than under normal conditions.

The investigations of the postoperative 17-ketosteroid excretion comprised 200 cases; in 140, i.e., about two-thirds, there was a significant rise, i.e., one exceeding 50 per cent. The increased excretion may commence in the course of the first four or five hours, but in most cases it does

corticoid excretion in the urine increases in the great majority of cases; in only few cases the rise in the excretion does not appear until the second or third day. The corticoid excretion falls to normal again between the second and sixth days, in the great majority of cases between the second and third. Our series includes fifty cases examined to the same extent as in the case of 17-ketosteroids (Figs. 4 and 5).

There was a significant rise in all our cases, i.e., a rise of more than 50 per cent. In a number of cases the corticoid excretion also showed a

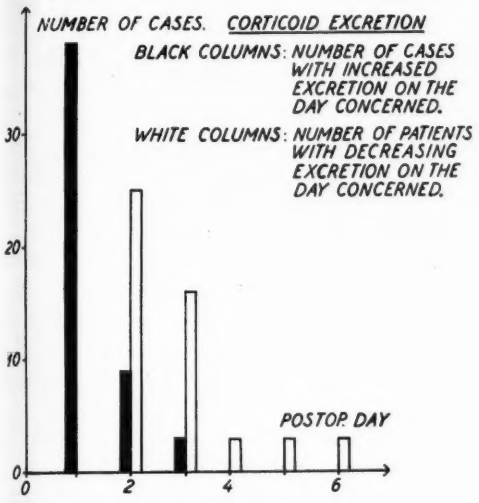


Fig. 4.

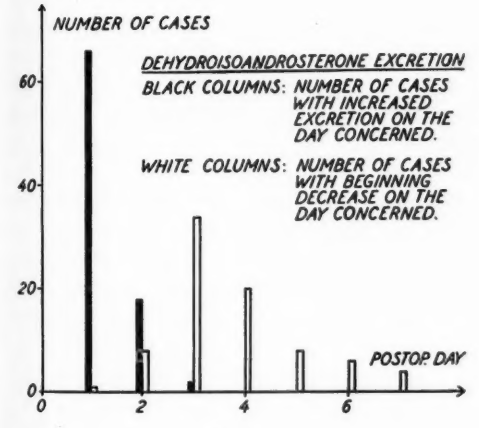


Fig. 6.

fall below the preoperative level after the postoperative initial rise. After a few days the corticoid excretion rises again to the normal level. Calculated in per cent the increase in the corticoid excretion is more pronounced than the corresponding rise in 17-ketosteroids, and the former rise more rapidly than the latter but also fall more rapidly. The corticoid excretion must be considered a more sensitive indicator of the adrenal cortex function than the 17-ketosteroid excretion.

The dehydroisoandrosterone excretion also proved to give a valuable expression of the adrenal cortex function following surgical inter-

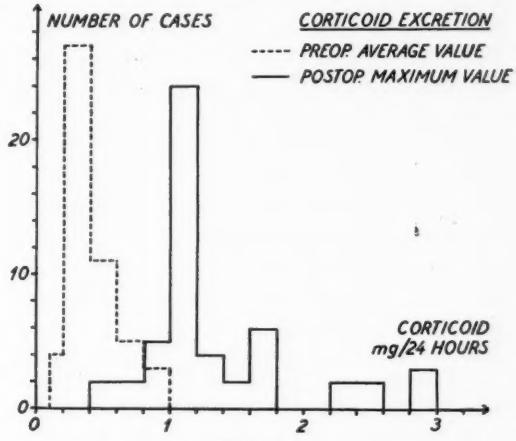


Fig. 5.

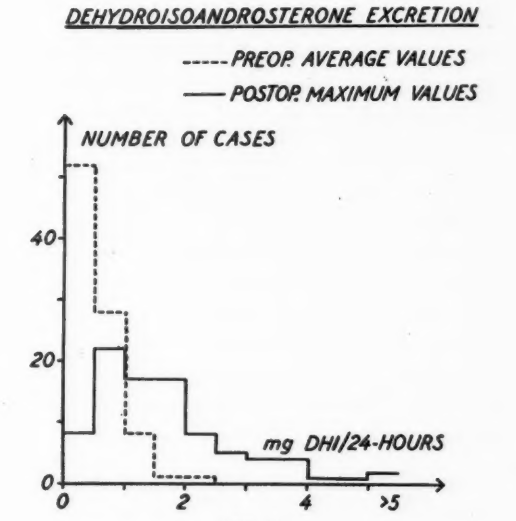


Fig. 7.

ference, and is apparently a sensitive indicator of this function. Dehydroisoandrosterone is also chiefly or exclusively produced in the adrenal cortex. The excretion was determined in ninety patients. In eighty-two there was a significant rise of over 50 per cent.

Figure 6 shows the time of onset and cessation of the increased excretion, and Figure 7 gives the preoperative and postoperative values.

In the great majority of cases the excretion rises during the first twenty-four hours, falling again

from the third to the seventh day, in a few cases below the preoperative level. As in the case of the corticoids the rise in per cent was more pronounced than that of the 17-ketosteroids.

We consider that the increased hormone excretion in the urine during the postoperative period which we have demonstrated in these investigations must be regarded as indicative of an increased adrenal cortex function. A comparison between the rises in the three hormone fractions whose excretion has been investigated shows that the significantly increased postoperative excretion amounted to 65 per cent, 80 per cent and 91 per cent for 17-ketosteroids, corticoids and dehydroisoandrosterone, respectively.

We have termed this increased adrenal cortex function the *primary, increased adrenal cortex function.*

Investigations on 17-hydroxycorticosterone in the blood performed by other authors in a smaller number of cases show results similar to those found in investigations on adrenocortical hormone excretion in the urine, and examinations of saliva which we have undertaken gave similar results.

In a number of cases a secondary rise occurred in the excretion of the three hormone fractions: *a secondary, increased adrenal cortex function.* The time of onset of this rise varied much; among the various causes that may produce such a rise we stopped to consider complications, which were the cause of the increased excretion in 50 per cent of the cases. In contrast with the primary rise the secondary rise occurred rapidly, in many cases before the complication could be ascertained clinically. The complications causing the secondary rise were of a serious nature.

In twenty cases we ascertained an increased excretion of the hormone fractions concerned beyond the ninth day; in all these cases the postoperative course was seriously complicated, and a few cases were fatal. We have termed this last-mentioned form of increased adrenal cortex function: *protracted, increased adrenal cortex function.*

Increased adrenal cortex function with the above-mentioned changes in the excretion of hormones is also associated with eosinopenia, which follows the changes in the hormone excretion; such a simple method of examination may thus indirectly afford a measure of the adrenal cortex function.

We performed eosinophil counts in 300 of the cases; in 288 we found a fall to under 50, and in 276 to zero. We attribute great importance to the almost constant fall to zero, as the great error in counting occurring in these counts is of no practical importance owing to this finding. The number of eosinophils during the postoperative period is thus a very sensitive indicator of increased adrenal cortex function.

The eosinopenia commences about half an hour after the beginning of the operation, and reaches maximum from four to five hours later. In uncomplicated cases it persists for one or two days, and then rises to the preoperative level on the average on the third day. The preoperative level is re-established on the seventh day at the latest. With regard to duration, the postoperative eosinopenia closely follows the increased excretion of hormones.

We examined in fifteen cases the possible effect of cortisone on the postoperative adrenal cortex function by observing the excretion in the urine of 17-ketosteroids and dehydroisoandrosterone. The dosage was 200 mg. of cortisone daily from three days before the operation to the sixth day after, with gradually decreasing doses to the tenth day. We found that cortisone reduces the excretion of 17-ketosteroids, and causes an even more pronounced reduction of that of dehydroisoandrosterone; but apparently cortisone only inhibits the increased postoperative excretion if there was a pronounced preoperative inhibition.

The increased adrenal cortex function is presumably the chief cause of the metabolic changes occurring during the postoperative period, but the renal and hepatic functions must also be included here in the pituitary-suprarenal axis system.

We performed a number of investigations on the potassium, sodium, chloride, protein, uric acid, creatinine and carbohydrate metabolism during the postoperative period in ninety-four patients (forty-three females and fifty-one males).

The potassium excretion in the urine was increased postoperatively in all patients, causing a negative potassium balance. In 84 per cent the excretion rose already within the first twenty-four hours after the operation, and in the rest of the cases on the second postoperative day. There was a relation as to time but no quantitative relation between the increased potassium excretion and the rise in the hormone excretion. In complicated cases, secondary increased or protracted increased potassium excretion was demonstrated, and in

these cases the serum potassium level became low.

The excretion of sodium and of chloride was lowered postoperatively in all patients examined.

The fasting blood sugar was determined in 419 patients (Fig. 8).

By comparing this series of patients admitted

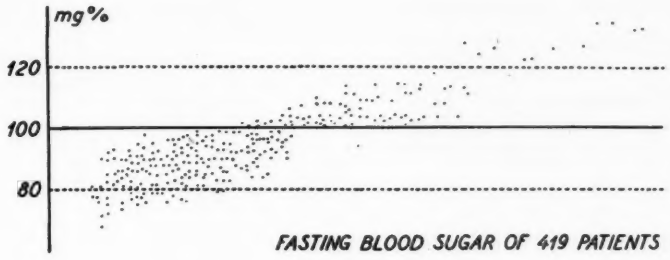


Fig. 8.

Relation as to time or quantity with the postoperative adrenocortical function could not be demonstrated either with regard to the excretion of sodium or that of chloride. The serum sodium level proved subject to small changes only, generally a fall, and no typical change in the serum chloride concentration occurred during the first few postoperative days.

The increased potassium excretion in the urine ceased in three days in 90 per cent of the cases, and in five days in all cases.

The sodium retention began about the second postoperative day and ceased about the fifth day.

The chloride retention began about the third postoperative day and ceased about the eighth day.

Both the sodium and the chloride retention begin later and persist longer than the increased renal potassium excretion.

The nitrogen excretion was increased, and a negative protein balance was found, lasting from three to ten days; increased uric acid and creatinine excretions were also observed, lasting from the second to the sixth or eighth postoperative day.

The carbohydrate metabolism during the postoperative period was examined by determinations of fasting blood sugar, glucose tolerance tests, demonstration of any glucosuria, and by examination of the insulin sensitivity. The activity of the pancreas can only be determined by these methods of indirect determination, which afford a possibility of deciding whether it is the pancreas or the other endocrine glands which determine the course of the carbohydrate metabolism postoperatively.

to the department with a series of normal individuals it appeared that the blood sugar level during the whole of the preliminary period of investigation was in the lower part of the normal interval. During the same period, and in the same series, the adrenocortical hormone excretion was also in the lower part of the normal interval.

Postoperatively we found a significant rise in the fasting blood sugar level, beginning during the first twenty-four hours, in about 80 per cent of the cases.

The blood sugar level began to rise immediately after the commencement of the operation, and continued to rise in the course of the day. The initial rise was rather abrupt and presumably caused by adrenaline. The rise then continues more slowly and is now presumably part of the actual stress reaction.

In most cases the hyperglycemia ceased about the third or fourth day, in some not until the tenth day (Fig. 9). Figure 10 shows the preoperative and postoperative blood sugar values. The former are average values, the latter maximal levels.

In contrast with the hormone excretion the fasting blood sugar did not fall below the preoperative level after the operation.

In the case of the fasting blood sugar level, too, examples of secondary rise and protracted rise were seen. In 66 per cent of these cases the cause of the rise were complications, and the secondary hyperglycemia occurred in many cases before the complication could be demonstrated clinically. We found a protracted rise in twenty-three cases, and the postoperative course was seriously complicated in all.

Intravenous glucose tolerance tests were performed in thirty-eight cases; twenty-six of these were operated on, and another glucose tolerance

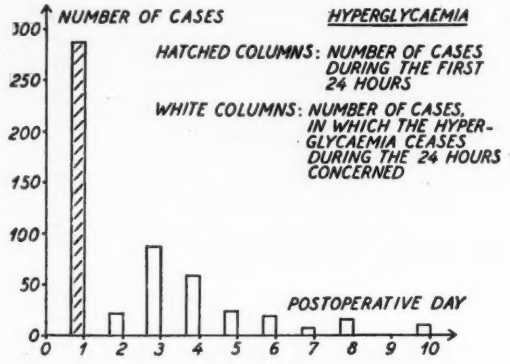


Fig. 9.

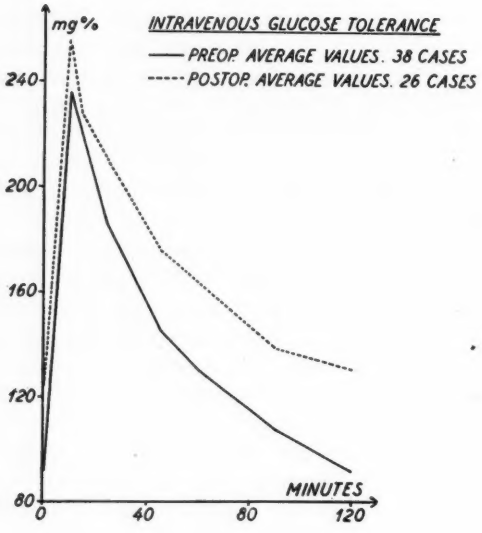


Fig. 11.

test was made after the operation. Figure 11 shows the average curve from preoperative and postoperative glucose tolerance test. It appears that the maximal blood sugar value was higher postoperatively than preoperatively, averaging 261 mg. per 100 ml. The fall was fairly parallel with the preoperative one, though somewhat slower and stopping at a higher level, so that the original value was not reached in two hours. The curve in the postoperative period rather suggests a lowered combustion and increased gluconeogenesis. From the third to the sixth postoperative day, most frequently on the fourth, the curve becomes normal again. In the case of complications the curve did not become normal until the complication had been obviated. Hyperglycemia

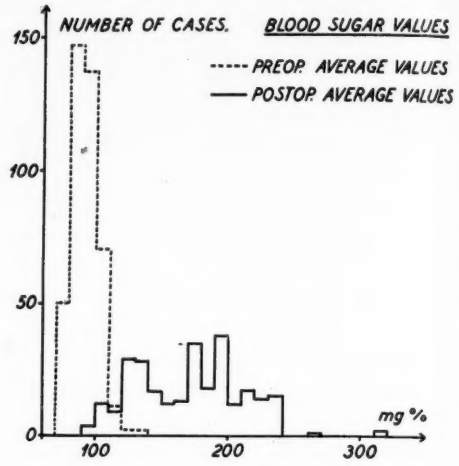


Fig. 10.

was present in all cases with abnormal glucose tolerance curves. Figure 12 shows the individual values.

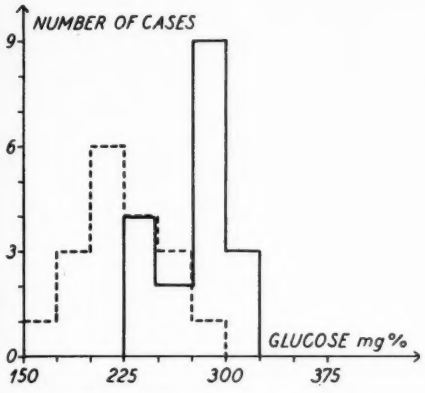
For the purpose of comparison, peroral glucose tolerance tests were performed in twelve cases (Fig. 13).

The preoperative and postoperative curves differed but slightly from each other, the latter rising and falling more slowly. The altered course of the curve in peroral tolerance tests is probably due to the changed conditions of resorption, transportation, and a changed regulation mechanism on the whole.

Our investigations thus showed a transitory change in a diabetic direction of the carbohydrate metabolism after operation, and suggest that glucose tolerance tests are more suitable as indicator of changes in the postoperative carbohydrate metabolism than determinations of the fasting blood sugar.

To obtain a knowledge of the factors causing the changed postoperative carbohydrate metabolism, we performed experiments on the insulin sensitivity before and after operation in sixty-three cases. In fifty-four of these cases the test (2 international units of plain insulin diluted to 2 cc. injected intravenously pre- and postoperatively) showed a change in the hypoglycemic reaction, as increased resistance to insulin occurred during the postoperative period; this was most pronounced

and most frequent on the second postoperative day, and then decreased during the days that followed, both in frequency and intensity. The



INTRAVENOUS GLUCOSE TOLERANCE.
--- PREOP. MAXIMAL VALUES
— POSTOP. MAXIMAL VALUES

Fig. 12.

gradual reduction of the resistance ceased about the sixth postoperative day.

Finally we examined whether the administration of cortisone influenced the fasting blood sugar levels before and after operation, but found no demonstrable changes in the carbohydrate metabolism; this may be due to the activity of the pituitary-suprarenal axis system exerting maximal influence.

The pituitary gland, the suprarenal glands, the ovaries and the thyroid gland all influence the carbohydrate metabolism. In the case of the pancreas our results do not permit definite conclusions, but apparently there is no increased insulin production, and any changes in the internal secretion of the pancreas do not influence the carbohydrate metabolism during the postoperative period.

Changes in the function of the parathyroids and of the gonads are hardly of any importance during the postoperative period.

The changes in the function of the thyroid gland during the postoperative period seem to suggest hyperfunction, as there is a rise in the metabolic rate postoperatively. We measured the metabolism in twenty-one patients in connection with operation. Nineteen showed a rise already during the first twenty-four hours, followed by a fall to the original level on the third postoperative day in some cases, whereas the rise persisted in a number of cases during the whole experimental period of nine days. Another rise

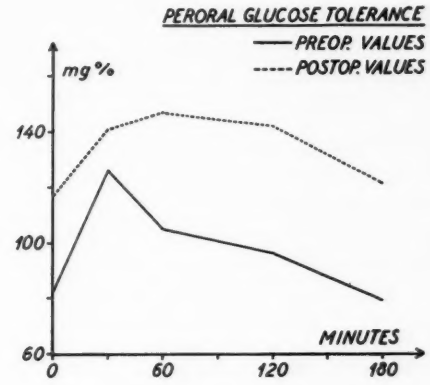


Fig. 13.

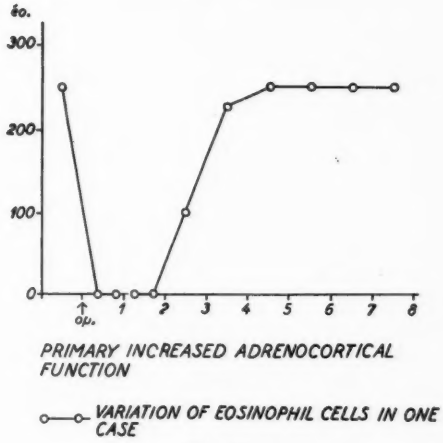


Fig. 14.

in the metabolism occurred if complications developed.

However, no direct conclusions with regard to the function of the thyroid gland can be drawn from the determinations of the metabolism only, but our investigations do not exclude the possibility of increased function. Other authors have demonstrated an antagonism between the adrenocortical and thyroid functions; to elucidate this antagonism we treated fifteen patients with cortisone before and after operation. We found that cortisone increased the energy transformation, so for this reason, too, no conclusions can be drawn with regard to the function of the thyroid from the determinations of the metabolism. Our investigations did not permit conclusions with

regard to the mentioned antagonism between the adrenocortical and thyroid functions.

As already mentioned, three types of increased adrenocortical function can be established on the basis of our investigations: primary, secondary, and protracted increased adrenal cortex function. These are of importance in practical clinical work, as they can be distinguished and identified by such a simple method of examination as an eosinophil count, which is very, but not too sensitive for this purpose (Figs. 14, 15 and 16).

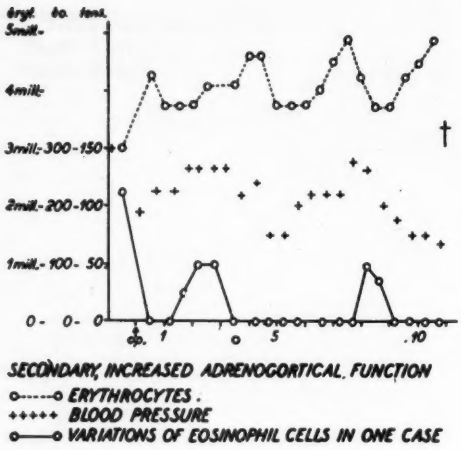


Fig. 15.

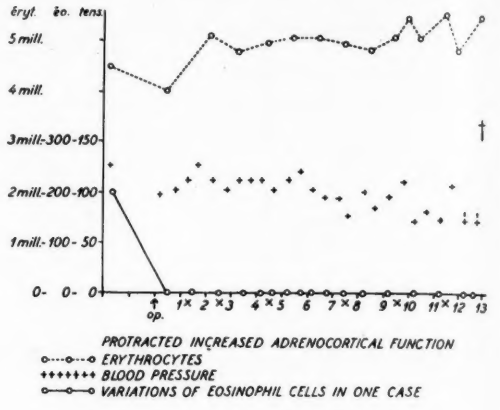


Fig. 16.

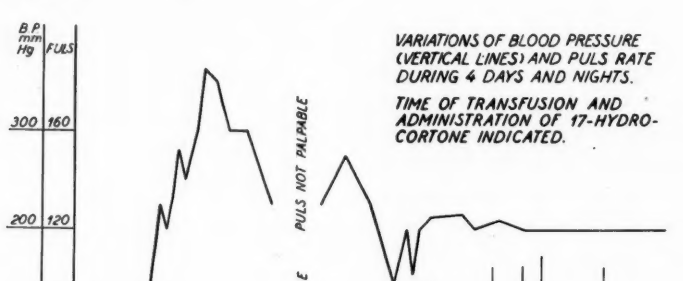


Fig. 17.

A secondary and a protracted increased adrenal cortex function occurs in conjunction with serious complications, and will often have set on before it has been managed to diagnose the complications clinically. It may therefore be of a certain diagnostic and possibly also prognostic importance to record the function of the adrenal cortex.

The changes in the metabolism during the primary increased adrenocortical function are clinically insignificant and require no treatment, whereas the occurrence of secondary and especially of protracted increased adrenocortical function requires observation of the water, protein and electrolyte metabolisms of the patients, because there is a risk of such pronounced deficits here that these may become dangerous if they are not properly dealt with. In cases with protracted hyperfunction we have observed a condition which we chose to call postoperative asthenia or metabolic shock: The patient is in a very poor state

(Continued on Page 380)

Cervical Traction and Other Physical Therapeutic Procedures for Pain about the Neck and Shoulders

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Rochester, Minnesota

A LARGE percentage of disabilities of the neck, shoulder and upper extremity are due to irritation or compression of the cervical nerve roots or some of the components of the brachial plexus. The physician may not be aware of the numerous possibilities for compression which exist in the region of the neck and shoulder. The most common disorders which produce signs and symptoms as a result of a mechanical irritation of the nerves are osteoarthritis of the cervical portion of the spinal column, protrusion of the cervical disk, whiplash injury and the so-called outlet syndrome.

Jackson[1] has proposed the term "cervical syndrome" to include most of the conditions which may be due to irritation of the cervical nerve roots or the cervical spinal nerves. Others have suggested the term "outlet or brachial plexus syndrome" for the conditions in which there is irritation or compression of the nerves of the brachial plexus.[2-4]

Pain in the region of the shoulder or in the arm may be due to lesions of the cervical portion of the spinal column as well as to lesions of the shoulder joint.[5] The most important point in differential diagnosis is the presence of local signs in the shoulder, that is, both active and passive limitation of motion, either diffuse or localized tenderness, or other signs of inflammation. In cases in which the shoulder joint is free of all these signs, the primary cause of the trouble usually is elsewhere. It must be remembered that a number of lesions of the chest may also cause pain in the shoulder and arm.

Anatomy

A knowledge of the functional anatomy of the cervical region is indispensable to the proper understanding of the mechanisms by which the cervical roots and plexus may sustain trauma by functional or anatomic variations from the usual.[6] The cervical portion of the spinal column allows a great range of motion in all directions. The seven cervical vertebrae enclose in their formed canal the eight cervical segments of the spinal cord and a variable portion of the upper thoracic segments.

The vertebra affords the greatest protection to the cord. The cord itself is relatively well protected in a bed of cerebrospinal fluid bounded by the arachnoid and dura mater. The junction of the second with the third cervical vetebra where the disk is thin is considered the weakest part of the spinal column, although this is not the part most liable to injury. The joints between the sixth and seventh cervical vertebrae allow the greatest mobility in the cervical region. However, the joints between the sixth and the fifth and between the fifth and the fourth cervical vertebrae are more vulnerable to injury than any other of these joints.[1] For example, when the neck is in hyperextension, the greatest strain is on the joints between the fourth and the fifth and stress is also on the joints between the fifth and the sixth vertebrae when the neck is in flexion.

A word of explanation is in order concerning the anatomic peculiarities of the cervical portion of the spinal column which has led to confusion in designating the level of neural, vertebral or intervertebral involvement. Since no disks are present between the occiput and the atlas or between the atlas and the axis, naming the disk involved by the number may be misleading. Thus, it seems desirable in specifying the level of involvement in the cervical region, especially when referring to a disk, to name the nerve involved or to specify the numbers of the adjoining vertebrae.[5]

According to Jackson,[1] there are small joints between the lateral portions of the bodies of the

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From the Section of Physical Medicine and Rehabilitation, Mayo Clinic and Mayo Foundation. The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

vertebrae which are called the "lateral intervertebral joints." Jackson considered them of great significance in the mechanism of irritation of cervical roots because of the close proximity to the nerve roots. The intervertebral foramina in the cervical portion of the spinal column are bony canals which are oval in shape and which have greater vertical diameter than anteroposterior diameter. An arthritic process may narrow the foramen so that the nerve root passing through may be irritated by the bony projections. The nerves leave the cord in the cervical region more nearly at a right angle than most lower nerve roots do, and they lie almost directly over the corresponding intervertebral disks. Therefore, a lateral protrusion of a cervical disk may compress a nerve root without damaging the cord.

This type of compression is now recognized as a rather frequent cause of brachial pain as first described by Semmes and Murphy.[7] In this situation it is easy to understand, as pointed out by Eaton,[2,3] how an increase of intrathoracic or intra-abdominal pressure may aggravate root pain because of the engorgement of the epidural venous plexus secondary to the interruption of the venous flow in the large vein. These engorged vessels press on or stretch the irritated nerve roots. The same author has stressed the accentuation in root pain which occurs during sleep. He attributed this to the lengthening of the spinal column that takes place when the body is in a horizontal position; this lengthening increases the pressure on the irritated nerve roots.

The seventh cervical nerve is considered to be the largest cervical root, and it supplies approximately the axis of the upper limb. When the limb is considered as a whole, it is readily seen that the fourth cervical and the first and second thoracic roots contribute to the sensory supply and there is definite overlapping. Thus, painful stimulation of a somatic region may cause reflex pain anywhere along the segmental distribution of the nerve or nerves which supply that particular region. For example, irritation of the dome of the diaphragm may cause pain in the arm on a reflex basis. Between the intervertebral foramina and the axilla are several other places where the nerve roots or plexus may be compressed by relatively minor anatomic variations. The underlying conditions may be grouped together and called the "outlet or brachial plexus syndrome."[2-4] These sites are the dorsal surface of the first rib, a rib attached to the seventh cervical vertebra, the posterior margin of an abnormally tight, hypertrophied scalenus anterior muscle, the region between the dorsal surface of the first rib and the inferior surface of the clavicle, and the region lying behind the pectoralis minor muscle and beneath the coracoid process. It has been known for a long time that the shoulder girdle descends during development. There is more descent in women than in men, and this, according to Todd,[8] would explain the greater frequency of symptoms due to brachial pressure in women than in men. It is interesting to note in this connection that most of the maneuvers used to test compression of the brachial plexus by the anterior scalenus muscle will also increase compression when other conditions of the outlet syndrome are present.

It is obvious, therefore, that many possibilities for compression of nerves exist in the neck and axillary region. Next, I shall consider in some detail several of these entities and describe the use of various physical therapeutic procedures in the treatment of these conditions with emphasis on home treatment.

Osteoarthritis

Osteoarthritis is a common cause of irritation of nerve roots. In 1925, Parker and Adson[9] provided one of the earliest descriptions of encroachment by hypertrophic spurs on the spinal foramina, causing irritation of the nerve roots, with symptoms referred to the region of distribution of some of the efferent portions of the particular nerves. Bony changes frequently occur in osteoarthritis of the cervical segment of the spinal column. These changes are found most often in the lower cervical vertebrae from which emerge the sixth, seventh and eighth cervical nerves. It is well to recall that these segments contain a portion of fibers which supply the pectoralis major and minor, the subscapularis and the deeper portion of the latissimus dorsi muscles. If the foramina from which these nerves emerge are narrowed, it can readily be understood how a patient could have pain over the pectoral region or the region of the shoulder and arm. Occasionally, a patient will have osteoarthritis of the cervical portion of the spinal column and complain of a dull ache largely confined to the region of a shoulder and arm. The patient may not complain of pain in the neck but he may have

noticed that certain movements of the neck, such as hyperextension, will produce pain in the region of the shoulder, arm or chest. However, when a patient complains of pain in the shoulder, in the arm or over the chest, it is well to keep in mind the fact that this pain may be due to osteoarthritis of the cervical vertebra.

The outstanding characteristic of this condition is the aggravation of all root symptoms by movement of the spinal column or by anything which tends to increase the pressure within the meninges. Sensory symptoms are common and almost all patients have pain or paresthesia. Physical examination may disclose decreased mobility of the cervical vertebrae, and crepitation is either audible or palpable over the back of the neck. Many of these patients complain of a grating sensation when they rotate their heads. Inasmuch as hypertrophic changes occur gradually over a long period and motion becomes limited, the nerve roots apparently sometimes accommodate to such foramina without symptoms; this may be an explanation of why many people in older age groups who have much osteoarthritis are not bothered by any difficulties except the gradual loss of motion of the neck. Narrowing of the intervertebral disks and hypertrophic changes about the joints seem to go hand in hand. It may be necessary at times to review a series of oblique roentgenograms to demonstrate encroachment on nerve roots in the foramina.

The treatment given in the Section of Physical Medicine and Rehabilitation includes the application of heat either by diathermy or baker for thirty minutes followed by sedative massage to the cervical, upper thoracic and shoulder regions of the involved side as well as the use of cervical traction. By preliminary application of heat and massage, the pain, muscular spasm and muscular tenderness can be modified so that the patient tolerates cervical traction much better. Our method is to apply traction, using a Sayre head sling with traction varying from 30 to 90 pounds, depending on the patient's tolerance and his ability to maintain relaxation of the cervical musculature during traction. The average pull is 60 pounds. While the traction is being applied, patients are instructed to shrug the shoulders and then relax; the physician then assists the patient to rotate his head slowly to the maximal range of motion two or three times. In some instances, the rotation is done entirely by the patient with the physician stabilizing the shoulders of the patient. All patients who are referred to our section for treatment are instructed in the home-treatment routine developed by Krusen.[10] The home treatment recommended consists of the use of a simple heat lamp or baker[11] followed by massage by some member of the family and then finally use of a simple home-treatment head sling (Fig. 1).

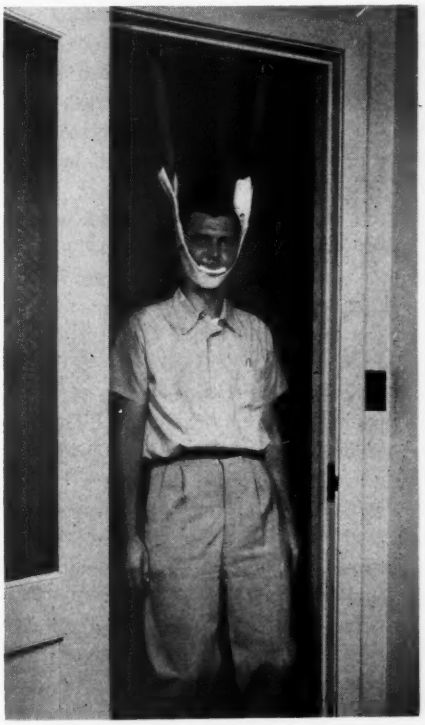

Fig. 1. Method of using home-treatment head sling. Someone should be with patient during this procedure to help him in and out of the sling.

Protruded Cervical Disks

Posterior or lateral bulging of a cervical disk or nucleus pulposus is now recognized as a frequent cause of brachial pain. Semmes and Murphy,[7] in 1943, pointed out that a laterally protruding disk markedly narrows the foramen and exerts pressure on the nerve root. If hypertrophy of the ligamentum flavum coexists, compression of the root will occur earlier. Cervical disks are constructed more delicately and are thinner than those in the lumbar region but they support much less weight. The disk between the sixth and seventh cervical vertebra is the most mobile and,

therefore, the most subject to trauma. According to Corbin,[6] this is the disk in the cervical portion of the spinal column which most commonly compresses the nerve root.

Pain is felt medial to the scapula on the affected side, sometimes over the pectoral region, and may extend from the shoulder down the arm into the forearm and, less often, into the hand and fingers. Irritation of the seventh cervical root usually causes parasthesia, less often pain, in one or more of the first three digits on the affected side, whereas compression of the sixth cervical root produces symptoms more commonly in the dorsum of the thumb. Lateral flexion of the hyperextended neck will often precipitate or aggravate the pain, especially if pressure is applied to the top of the head.

Traction on the cervical portion of the spinal column may give prompt relief if a protruded disk is pressing on a cervical nerve. The pain is aggravated by coughing, sneezing or straining and usually is accompanied by splinting of the neck muscles, with tilting of the head away from the side affected. Pressure over the lateral cervical spines or transverse processes may be painful. Objective sensory changes may be detected in the hand corresponding to the paresthetic zone. Weakness of specific muscles is infrequent but may be detected in the biceps or triceps. Similarly, the biceps or triceps reflexes may be diminished or absent. X-ray examination of the cervical portion of the column in the presence of a protruded disk usually will disclose a loss of the normal curve with narrowing of the interspaces. Patients with this condition who are referred to the Section of Physical Medicine and Rehabilitation are given essentially the same treatment as that for cervical osteoarthritis.

It is our impression that intensive vertical traction for short periods is more effective than cervical traction applied with the patient in bed in the supine position.

Whiplash Injuries

Gay and Abbott[12] pointed out how frequent automobile accidents occur as a result of a rear-end collision and that a whiplash injury is the common sequela. Patients who have sustained an injury of the whiplash type from such an accident or from any other cause have the symptoms and findings characteristic of sprain of the neck. These consist of pain, limited motion, and spasm and tenderness of neck muscles. Movement or jarring of the neck makes the symptoms worse and many of these patients tend to support their head and neck with their hands. In the series reported by Gay and Abbott,[12] 26 per cent of the patients with whiplash injuries had persistent cervical radiculitis with pain and paresthesia extending into one upper extremity or alternating in both extremities, indicating protruded intervertebral disk. Other of their patients were described as having hypertrophic arthritis of the cervical portion of the spinal column and some had subluxations of the lower cervical vertebrae. Many of the patients who are seen at the Mayo Clinic with this condition have had their symptoms for weeks or even months, and recently we had a man who had worn a neck brace for three years.

Gay and Abbott[12] pointed out that constant wearing of a neck support results in atrophy of the cervical muscle and adds the complicating factor of weakened cervical muscles to the patient's problem. These patients are benefited by the use of a cervical collar in the early stages of this disorder but it seems important to make sure that the patient gradually discontinues its use after the neck limbers up.

The program in our department has been essentially the same as that described for the treatment of osteoarthritis of the neck or for the treatment of cervical disks. Since many of these patients have marked limitation of cervical motion, a much longer period of treatment is required in order to bring about improvement in the range of motion. Furthermore, since some of these patients have weakness of the cervical muscles, we add strengthening exercises to their program.

Outlet Syndrome

All of the conditions producing the outlet syndrome have a similar pattern of symptoms, namely, that of compression of the brachial plexus. The variations of terms for this syndrome have arisen from the numerous explanations of the underlying pathology. The etiology is confusing because so many anatomic deviations have been found in the structures of the neck. The correction of each by surgical means has generally brought relief.

Adson's maneuver is helpful in the diagnosis. It is performed as follows: Radial pulses are compared for difference in volume, since the outlet syndrome is usually unilateral. The patient with

the arm in a dependent position is asked to turn his face toward the side being examined, to take a deep breath and to hold it, while hyperextending the neck. The pulse volume on the side being examined decreases greatly or fades out and usually the patient's symptoms are aggravated. In the majority of cases, according to Eaton,[2] none of the structural deviations in the neck and shoulder usually described for any one of the conditions in the group can be demonstrated. As a result, treatment of the outlet syndrome has been varied. Also, symptoms have recurred following operation for scalenus anticus syndrome, cervical rib or one of the other syndromes. All of these patients will have pain and paresthesia in the upper extremity. The pain almost never resembles that produced by compression of a nerve root in that there is no strain aggravation. The symptoms of the hyperabduction syndrome are caused by pressure on the vessels and nerve trunks which pass posteriorly to the pectoralis minor and beneath the coracoid process when the arm is elevated as during sleep.

When the etiology of these various types of compression syndromes of the brachial plexus is considered, attention must be paid to the details of posture which might play a part in producing symptoms. It has been pointed out by Todd[8] that with a gradual descent of the shoulders during childhood and in early adult life, the brachial plexus is stretched over the first rib and symptoms may result without any real anatomic variation.

In the Section of Physical Medicine and Rehabilitation at the clinic we have formulated a definite program of treatment for these patients which consists of heat and massage and corrective exercises for the neck and shoulders. Progressive resistance exercises are used to strengthen the shoulder elevators and retractors and, in addition, posture training is given.

Summary

A large percentage of the disabilities of the neck, shoulder and upper extremity which are due to irritation or compression of cervical nerve roots or some of the components of the brachial plexus are amenable to physical therapy.

Osteoarthritis of the cervical vertebrae with irritation of the nerve roots, protruded cervical disk or whiplash injury is treated with heat applied locally, sedative massage and traction of the cervical portion of the spinal column by the aid of a Sayre head sling. Heat and sedative massage to neck and shoulder girdles and special exercises to improve posture and to strengthen the shoulder retractors and elevators are helpful in relieving the symptoms of the outlet or brachial plexus syndrome. Patients can be instructed to carry out these procedures at home under the supervision of the physician.

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Arteriography as an Aid to Diagnosis

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VISUALIZATION of the arterial tree of an extremity was first carried out many years ago, on an amputated arm and hand. However, it was not until 1923 that visualization of the arterial tree of an extremity of a living person was accomplished. Since that time arteriography has slowly gained favor as a help in delineating the vascular tree of an involved extremity or set of vessels.

Fig. 1. Materials necessary for percutaneous arteriography.

The basic objectives of making an arteriogram concern demonstration of five aspects of the vascular system. These are: (1) the anatomic aspects, whether they are abnormal or normal, (2) local changes in the vessels, such as occur in arteriosclerosis or thromboangiitis obliterans, (3) occlusion of major vascular trunks, (4) the presence of aneurysms, and (5) the presence of arteriovenous fistulas.

There are two methods of visualizing the vascular tree of an extremity by means of radiopaque material. These are the open injection method and the percutaneous injection method. In the open method the artery is opened down upon, and under direct vision the needle with its accompanying catheter is introduced into the vessel and injection is carried on from that point. In the percutaneous method the vessel is punctured by entering through the skin and the catheter is fed into the artery, the needle subsequently being withdrawn, leaving the catheter in place. Then the injections are carried out as in the open method.

The materials necessary to produce a suitable arteriogram are outlined in Figure 1. The two metal mixing bowls are necessary for dilution of the contrast medium (solution of urokon sodium) from 70 to 35 or 50 per cent, whichever is necessary for use in the extremities. We use No. 14 needles, either 2 or 2½ inches long, complete with stylets. Catheters of the size of a No. 18 needle will easily pass through the bore of such needles. Syringes of 20 cc. and 50 cc. capacity, catheters and hemostats used to clamp the catheters also are shown in the figure.

The technique of injection is relatively simple. After the artery has been entered and it is certain that a free flow of blood back into the catheter has been obtained, 50 cc. of the solution of urokon is injected. The first 15 to 20 cc. is injected slowly, to fill the vascular tree and to perfuse the collateral vessels, after which the remainder of the dye is injected as rapidly as can possibly be done. The roentgenogram is made during injection of the last 10 cc. of the solution. The x-ray film is centered over the portion of the extremity which it is wished to visualize. If it is desired to visualize more material than can be accommodated within the standard x-ray film measuring 14 by 17 inches, two x-ray plates can be used or two separate x-ray exposures can be made at dif-

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Fig. 2. (*a*) Paget's disease of the upper part of the tibia. (*b*) Fibrosarcoma of the thigh.

ferent levels by the use of two separate injections. The exposure time, so far as the roentgenogram is concerned, usually is 2½ to 3 seconds, but may vary as to actual need. It is prudent to make a check roentgenogram, so that the technician will have the proper exposure time and other factors on the machine prior to injection of the dye.

The figures illustrate several cases in which an arteriogram has been of help. Figure 2*a* is an arteriogram made for a man who had a pulsation in the upper part of the left tibia. It was suspected that he had Paget's disease. The arteriogram revealed that a considerable amount of the dye had lodged in the upper part of the tibia. The arteriogram thus indicated that the man probably had many minute arteriovenous fistulas.

Figure 2*b* shows a mass in the mid-part of the thigh. This mass subsequently proved to be a fibrosarcoma of the thigh. The arteriogram demonstrates considerable abnormal vascular channels, a finding which is helpful in arrival at the diagnosis of a malignant tumor.

Figure 3*a* is an arteriogram of a pulsating mass in the mid-portion of the femoral area. The arteriogram demonstrates an aneurysm of the right superficial femoral artery.

Figure 3*b* is an arteriogram of a pulsating mass in the popliteal space. This mass was thought to be a popliteal aneurysm. The arteriogram, which was made to delineate the aneurysm and to sustain or disprove the diagnosis, revealed that the lesion was not an aneurysm at all, but rather, a

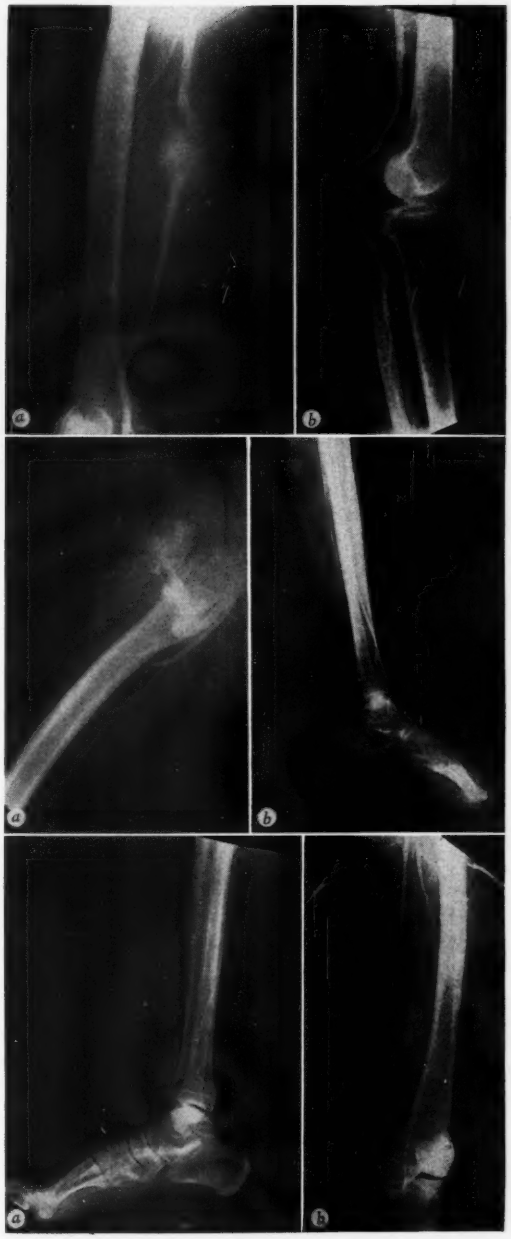

Fig. 3. (top) (*a*) Aneurysm of the superficial femoral artery. (*b*) Popliteal synovial cyst.

Fig. 4. (center) (*a*) Cavernous hemangioma of the upper part of the thigh. (*b*) Occlusion of the dorsalis pedis artery.

Fig. 5. (bottom) (*a*) Occlusion of the posterior tibial artery. (*b*) Segmental occlusion of the superficial femoral artery.

tumor near the posterior surface of the knee joint which had displaced the popliteal artery posteriorly. Surgical exploration proved this lesion to be a synovial cyst or ganglion cyst of the popliteal space.

Figure 4*a* is an angiogram of a palpable mass in the upper part of the thigh. This lesion at palpation imparted the sensation of a water-filled sponge. The angiogram showed it to be a cavernous hemangioma.

Figure 4*b* depicts an unhealed ulcer on the dorsum of the foot. The patient was a diabetic woman, and the roentgenogram shows that previously she had lost all her toes. The unhealed ulcer considerably complicated the management of the diabetes, and an arteriogram was made to visualize the vascular tree of this extremity. Note that the arteriogram has revealed occlusion of the dorsalis pedis artery.

Figure 5*a* shows the foot of a man who had an unhealed ulcer beneath the external malleolus and considerable ischemia of the foot. An arteriogram was made to visualize the vascular tree of this involved extremity. It revealed a diseased superficial femoral artery and an occluded posterior tibial vessel.

Figure 5*b* is an arteriogram of the thigh of a man who had intermittent claudication and pain in the calf and thigh, ascending into the buttocks, after he walked more than a half-block. Arteriography of this involved extremity disclosed segmental occlusion of the superficial femoral artery.

In conclusion, it is believed that these examples demonstrate the value of an arteriogram as an aid to diagnosis, as a distinguishing technique in diagnosis and as a means of delineating vascular disease.

FUNCTION OF ENDOCRINE ORGANS

(Continued from Page 372)

with slow cerebration, dehydration and potassium deficiency; the acid-base balance is disturbed, the blood volume reduced, et cetera, but generally no other circulatory disorders are observed.

The question arises whether adrenocortical hypofunction develops in such a condition. This question is difficult to answer. We have in no instance seen hormonal and physiopathological disturbances suggesting insufficiency, and especially in no case of protracted rise in the adrenocortical function observed the development of hypofunction. On the other hand, there may be a relative or partial adrenocortical insufficiency in such cases.

Observations which we made in five cases with severe, postoperative circulatory insufficiency, with a fall in blood pressure to 60 to 50 mm. Hg, treated with 17-hydrocorticosterone (400 to 500 mg. per twenty-four hours intravenously), seem to support the supposition of the possibility of partial or relative adrenocortical insufficiency.

It was impossible in these cases to obtain an essential rise in blood pressure with blood transfusions. In all five cases the administration of 17-hydrocorticosterone caused a fall in pulse rate and a rise in blood pressure to over 100 mm. Hg. In addition there was a material improvement in the general health, both psychically and somatically. However, it was managed only to take one of the four patients over the crisis.

Figure 17 shows the course in one patient. The blood pressure and the pulse variations are shown in relation to the administration of blood and 17-hydrocorticosterone (preparation hydrocortone), respectively.

Finally, I should like to emphasize that the best prophylaxis of securing satisfactory operative results is a thorough preoperative treatment and good surgery.

Minnesota Maternal Mortality Study for 1953

IN this report, all of the known maternal deaths occurring from April 1, 1953, through March 31, 1954, have been evaluated, and these data will be called "The 1953 Survey." Previously published committee reports for the years 1941-1942, 1950, 1951, and 1952 will be used for comparisons. The details of methods and criteria of the study have been published. All known deaths occurring during pregnancy or within three months after termination of pregnancy are included for the study. Deaths due to chorioepithelioma are included in the study for the year in which they occur.

TABLE I. MATERNAL DEATHS IN MINNESOTA STUDIES FOR 1941, 1950, 1951, 1952 AND 1953

Year	Number Deaths	Number Births	Gross Mortality Rate per 1,000 Live Births†	Maternal Mortality Rate per 1,000 Live Births‡	U.S. Maternal Mortality Rate per 1,000 Live Births
1941	112	55,293	2.03	1.68	2.8 *
1950	68	76,074	0.89	0.59	0.83
1951	57	80,099	0.71	0.45	0.75
1952	58	78,990	0.73	0.42	0.68
1953	59	79,362	0.74	0.42	0.62**

*Average for 1941 and 1942
**Unofficial
†Including all maternal deaths from whatever cause.
‡Excluding deaths considered as non-obstetrical on the basis of the definitions set down by the United States Bureau of the Census, but using the information obtained from detailed study of each case. This latter adds to the number of so-called "obstetric" deaths some of which would be considered "non-obstetric" on basis of death certificate information alone.

As previously emphasized, the usual method of using death certificate data exclusively for compilation of statistics on maternal deaths is inaccurate and inadequate. All of the deaths in pregnant or parturient women are used to calculate the gross maternal mortality rate for the state, and the corrected number, listing only the "obstetrical deaths," is used for the Minnesota Maternal Mortality rate. These rates for the years of the study appear in Table I. The 1953 figure compares favorably with those of recent years and reflects continued excellence of obstetric care afforded by Minnesota physicians. Most of the results for 1953 as presented in the subsequent tables are essentially the same as that for 1952. The differences as noted in comparing the 1953 figures with the 1950-1952 averages are due to higher rates for the first two of those years.

TABLE II. SOURCE OF REPORT OF MATERNAL DEATHS

	1950-52		1953	
	Number	Per Cent	Number	Per Cent
Cases found from death certificate (with or without prelim. reports from physicians, hospitals, etc.)	135	73.8	43	72.9
Sources other than death certificate	48	26.2	16	27.1
Cross match of death with birth certificates— 11=18.6% Others 5= 8.5%				

Table II shows the sources of reports of maternal deaths. About the same per cent of additional cases was found this year by means other than standard reporting. Cross matching death certificates with birth certificates turned up eleven additional cases, and other sources such as reports from hospitals and physicians brought five more to our attention, for a total of 27 per cent of the cases. In this group of sixteen, two patients died postpartum of rheumatic heart disease, two depressed patients committed suicide, and one died of pulmonary embolism on the eighteenth day after delivery. This serves to emphasize the type of case which would be missed using death certificate information only. The others in this group died of "non-obstetrical" causes. To make the study all inclusive, the State Department of Health requires a report within three days of death of all patients dying during pregnancy or in the three months postpartum period. These should be reported regardless of whether or not the death was thought to be "obstetrical." Because of failure to comply with this requirement, there are probably some cases which are still being missed for study when death occurs before delivery or in pregnancies delivered before the twentieth week when a birth certificate is not required.

Fifth annual report of the Minnesota Maternal Mortality Study Committee, subcommittee of the Maternal Health Committee of the Minnesota State Medical Association.

TABLE III. ANALYSIS OF DEATH CERTIFICATES

	1950-52		1953	
	Number	Per Cent	Number	Per Cent
Death certificates complete and correct in all details	57	31	22	38
Death certificates correct in all details reported	54	30	16	27
Death certificates incorrect	72	39	21	35
Death certificates incorrect as to cause of death	(52= 28%)		(14= 23.8%)	
Total maternal deaths	183	100.0	59	100.0

Inaccuracies of Death Certificates

The data for maternal mortality reports are generally based on death certificate report only. The inadequacy of this is pointed out above. The inaccuracy of this has been previously reported and is again evident as shown in Table III. In the 1953 study only 38 per cent of the death certificates were complete and correct in all details. Thirty-five per cent were incorrect in some detail. Twenty-seven per cent were incomplete. In 23.8 per cent the cause of death was incorrect. This represents a considerable error which can be detected only by individual study of each case. The reasons for the errors are much the same as in the past. In three cases a positive cause of death was reported when actually the cause was unknown and should have been reported as such. One patient whose death was reported as being due to "premature separation of the placenta and Rh factor" died six hours after delivery of inadequately treated postpartum hemorrhage. One whose death was attributed to acute toxemia died from the administration of Rh incompatible blood. Another patient who died of puerperal infection was signed out as dying from massive pulmonary edema. These suffice to demonstrate again the major inaccuracies. A multiplicity of errors in reporting, or failure to report minor details as required on the death certificates reflects carelessness in the filling out and signing of these documents.

Results of the 1953 Survey

The number of preventable maternal deaths in 1953 was 15 or 25.4 per cent of the total. This is more than the 17.2 per cent reported in 1952 but compares favorably with the 30.6 per cent average for the 1950-1952 period (Table IV). This certainly reflects a considerable improvement over the 73.2 per cent of preventable deaths as reported in the 1941 study. The total number of "obstetric" deaths is the same as for last year but percentagewise shows improvement over the averages of the previous years (Table V).

TABLE IV. PREVENTABILITY OF MATERNAL DEATHS IN MINNESOTA

	1941		1950-52		1953	
	Number	Per Cent	Number	Per Cent	Number	Per Cent
Preventable	82	73.2	56	30.6	15	25.4
Not preventable	27	24.1	124	67.8	44	74.6
Not determinable	3	2.7	3	1.6	0	

TABLE V. OBSTETRIC AND NON-OBSTETRIC DEATHS

	1941		1950-52		1953	
	Number	Per Cent	Number	Per Cent	Number	Per Cent
Obstetric deaths	93	83.0	118	64.5	33	55.9
Non-Obstetric deaths	19	17.0	65	35.5	26	44.1

TABLE VI. TYPE OF DELIVERY

	1950-52		1953	
	Number	Per Cent	Number	Per Cent
Undelivered	65*	35.5	19**	32.2
Spontaneous	53	29.0	16	27.2
Operative	67	35.5	24	40.6
Indicated	43		21	
Not indicated	24		3	

*Includes 7 delivered by postmortem section.
**Includes 3 delivered by postmortem section.

Table VI shows the number of operative deliveries for this year. There were twenty-four as compared with twenty in 1952. Those in which the operation was not indicated remain the same at three (12.5 per cent). Improvement in this direction is evident when one compares this to a total of 36 per cent unindicated operations in the 1950-1952 combined series. Three post-mortem sections were done and in all of these the fetuses were stillborn. This brings to a total of fifteen the post-mortem sections appearing in this study in the past four years, and in none was a live fetus produced. Another patient reported as having a post-mortem section in the 1953 study was actually sectioned in the agonal state following a cerebral vascular accident. This fetus survived for thirty-

TABLE VII. SEROLOGIC TEST FOR SYPHILIS

	1941		1950-52		1953	
	Number	Per Cent	Number	Per Cent	Number	Per Cent
Unregistered	18		31		11	
Not obtained	64	68.1	83	54.6	21	43.8
Obtained	30	31.9	69	45.4	27	56.2

TABLE VIII. RH DETERMINATION

	1950-52		1953	
	Number	Per Cent Registered Patients	Number	Per Cent Registered Patients
Unregistered	31		11	
Not obtained (Registered patients)	48	31.6	10	20.8
Obtained at hospital	30	19.6	8	16.6
Obtained early in pregnancy	73	48.1	29	60.5
No data	1	0.7	1	2.1
Totals	183	100.0	59	100.0

TABLE IX. CONSULTATIONS

	1941		1950-52		1953	
	Number	Per Cent	Number	Per Cent	Number	Per Cent
None	63	56.2	79	43.1	19	32.2
Indicated			39		6	
Not indicated			40		13	
Adequate	10	9.0	69	37.7	33	56.0
Inadequate	39	34.8	35	19.2	7	11.8
Totals	112	100.0	183	100.0	59	100.0

TABLE X. PELVIC MEASUREMENTS

	1941		1950-52		1953	
	Number	Per Cent Registered Patients	Number	Per Cent Registered Patients	Number	Per Cent Registered Patients
Unregistered	18		31		11	
Not measured or incompletely measured	77	81.9	97	63.8	27	56.3
Measured	17	18.1	55	36.2	21	43.7

six hours. Eight patients who died were delivered by cesarean section. Only one cesarean section was considered as not being indicated. The other unindicated operative deliveries were as follows: in one, the patient was delivered by version and extraction and died of hemorrhage due to uterine rupture; another was delivered by accouchement forcé followed by version and extraction. This patient, who had a placenta previa, died of hemorrhage and shock. This unindicated procedure continues to contribute to avoidable maternal mortality despite the hope that it was a thing of the past when none were reported in the 1952 study.

Table VII shows the number of patients who had serologic tests for syphilis during early pregnancy. This figure of 56.2 per cent is another which is essentially the same as for 1952 but it reflects improvement over the averages for the 1950 to 1952 series. A similar circumstance is shown in the use of Rh tests early in pregnancy (Table VIII). Here the per cent obtained early in pregnancy is about the same for 1952 but again ahead of the three-year average.

The trend toward more frequent use of better consultants is shown in Table IX. Inadequacies persist due to failure to use the better trained men in some communities as consultants and in asking for consultation too late for it to be of value. There were six cases in which consultation was indicated, consultants being available but not used. In four of these the death was considered preventable, and adequate consultation would have been most helpful in trying to change the course of events.

The figures presented in Table X still show considerable neglect of pelvic mensuration in the cases studied. Some of this related to failure to recheck pelvimetry in multiparous patients who present themselves for prenatal care for the first time in the given office. In other cases the measurements were said to have been done but not recorded. The measurement of the diagonal conjugate, transverse diameter of the outlet, and the palpation of the ischial spines and sacrum require a minimum of effort and should be recorded in all cases. These measurements, with the palpation of the internal pelvic contours, are sufficient to pick up the candidates for x-ray pelvimetry. Despite lack of pelvic mensuration there were no deaths in the 1953 study due to cephalo-pelvic disproportion.

The adequacy of prenatal care is summarized in Table XI. Only 16.7 per cent of the patients in the 1953 study had adequate prenatal care as

TABLE XI. PRENATAL CARE

	1941		1950-52		1953	
	Number	Per Cent	Number	Per Cent	Number	Per Cent
Adequate	2	1.9	31	20.2	8	16.7
Faulty, contributory	55	52.9	29	19.0	5	10.3
Faulty, not contributory	47	45.2	93	60.8	35	73.0
None	8		30		11	
Totals	112	100.0	183	100.0	59	100.0

TABLE XII. CARE IN LABOR AND DELIVERY

	1941		1950-52		1953	
	Number	Per Cent	Number	Per Cent	Number	Per Cent
Adequate	7	7.8	55	38.7	18	44.0
Faulty, contributory	61	67.8	53	37.3	10	24.4
Faulty, not contributory	22	24.4	34	24.0	13	31.6
None	22		41		18	
Totals	112	100.0	183	100.0	59	100.0

compared to 21.3 per cent in 1952. The present figure is also lower than that of the three-year average as shown in the table. The number of patients having faulty prenatal care contributing to the patient's death was the same in each of the last two years. With respect to care in labor and delivery, the 1953 study compares favorably with the 1950-1952 average but the 24.4 per cent who had faulty delivery care contributory to the patient's death suffers by comparison with the 8.9 per cent reported in 1952. The figures for post-partum care show a trend toward improvement.

The frequency with which adequate autopsies were obtained has increased. 49.2 per cent is an excellent level in any group of patients, but can be improved (Table XIV). Thirty-four per cent failure to request autopsies is much too high. Among the reasons given for this is the lack of an available pathologist. Arrangements can be made for a pathologist to do these autopsies if the State Department of Health is notified of the death. Autopsy is certainly indicated in the cases in which the cause of death is unknown. There are others in the study in which the cause of death was probable but not proven, so that here too the usefulness of adequate autopsy is apparent.

TABLE XIII. POST PARTUM CARE

	1941		1950-52		1953	
	Number	Per Cent	Number	Per Number	Number	Per Cent
Adequate	4	4.8	48	43.6	21	58.4
Faulty, contributory	67	79.8	51	46.4	9	25.0
Faulty, not contributory	13	15.5	11	10.0	6	16.6
None	28		73		23	
Totals	112	100.1	183	100.0	59	100.0

TABLE XIV. AUTOPSIES

	1941		1950-52		1953	
	Number	Per Cent	Number	Per Cent	Number	Per Cent
Obtained, adequate	9	8.0	71	38.8	29	49.2
Obtained, inadequate	27	24.1	6	3.3	1	1.6
Not requested	55	49.1	70	38.3	20	34.0
Permission refused	21	18.8	36	19.6	9	15.2
Totals	112	100.0	183	100.0	59	100.0

TABLE XV. PRIMARY CAUSES OF MATERNAL DEATHS IN MINNESOTA, 1953 STUDY

Cause of Death		Number	Per Cent
Toxemia		8	13.5
Hemorrhage		4	6.8
Transfusion accidents		4	6.8
Heart disease		4	6.8
Infection		4	6.8
Suicide		2	3.4
Pulmonary embolism		2	3.4
Amniotic fluid embolism		1	1.7
Pituitrin shock		1	1.7
Acute pulmonary edema		1	1.7
Anesthesia (hexylcaine HC1)		1	1.7
Diabetic acidosis		1	1.7
Non-obstetrical complications		26	44.1
Bulbar poliomyelitis	6		
Intracranial hemorrhage	4		
Neoplasms	4		
Peritonitis	3		
Trauma	4		
Unknown	3		
Meningitis	1		
Lupus erythematosus	1		
Totals		59	100.1

Causes of Death

The causes of death in the 1953 study are listed in Table XV. There were thirty-three "obstetrical" deaths, which compares favorably with the thirty-three "obstetrical" deaths in the 1952 study. Toxemia accounts for the largest number of deaths as it did in 1950. In 1951 and 1952 deaths due to hemorrhage headed the list. Two of these eight patients had not registered for pre-

natal care. One had a fulminating pre-eclampsia upon arrival at her local hospital and convulsed one and three-fourths hours later. She had signs of a cerebral vascular accident and remained comatose until death. The other unregistered patient had a severe arteriolosclerotic toxemia. She was hospitalized immediately after her first office visit at the thirty-sixth week, at which time she had no symptoms. Soon thereafter she had a convulsion, following which she remained comatose until death two hours later.

Another patient at the twenty-ninth week of gestation developed pre-eclampsia which was well controlled during nine days of hospitalization. At the end of that time she developed a fulminating pre-eclampsia which was treated medically. A convulsion occurred five hours after this change and she remained comatose until death. There was failure here to interrupt the pregnancy with the onset of the fulminating pre-eclampsia. Another patient developed eclampsia at home the twenty-eighth week of pregnancy. She responded well to medical management after hospitalization. Two days later she had increasing symptoms of toxemia for a time, was better the next day, and suddenly worsened and died on the fourth day after the onset of eclampsia. This pregnancy should have been interrupted during the time when the eclampsia was under control. Two patients with arteriolosclerotic toxemia developed ablatio placentae. One of these had renal failure prior to the onset of the symptoms of placental separation, was treated by hysterotomy, and remained anuric until death eight days later. The other had the findings of placental separation, was treated by cesarean section and was thereafter anuric until death. There was no data regarding renal output prior to the operation.

The seventh patient in this group had a long history of pre-eclampsia. She was admitted to the hospital at term in anticipation of interruption of the pregnancy by induction or section. During that night she developed signs of fulminating pre-eclampsia which were overlooked for several hours. Then, during the convulsive phase, she was delivered by cesarean section. The convulsions continued at frequent intervals until her death. Here an opportunity at earlier interruption was missed. It was unwisely undertaken before the convulsions were under control. The last of these patients had her initial convulsion while she was bathing in a tub. The eclampsia was satisfactorily managed but she died of pneumonia due to the aspiration of the soapy water. Three of these eight were considered to be preventable deaths.

Of the patients who died of hemorrhage, one was seen by a physician at home early in labor. He failed to diagnose either the pregnancy or labor. He was called to see her again three days later, at which time she was found to be dead. Delivery occurred some hours before but the placenta was still in place. The most likely cause of death was shock due to hemorrhage associated with retained placenta. Another patient had intrapartum bleeding of minor amount and of undiagnosed origin. She died about six hours after delivery from hemorrhage due to untreated postpartum uterine atony and delayed, inadequate blood replacement. A third in this group was delivered by an unindicated version and extraction, done twelve hours after membrane rupture. She suffered a uterine rupture and promptly died of untreated blood loss. The fourth had a placenta previa. She was delivered by accouchement forcé with lacerations of the birth canal. After this she continued to bleed profusely and remained in shock until death. Aside from the faulty delivery procedure she had inadequate and delayed blood replacement. All of these hemorrhage deaths were considered preventable.

A reduction in the total number of deaths due to hemorrhage is not without its price, as shown by the occurrence of four transfusion accidents in the present year's study. The value of adequate blood replacement is without question. However, the dangers associated with the administration of blood should be re-emphasized. In one instance a patient was receiving a blood transfusion following an abortion. The flow of blood was rather slow and air pressure was applied to the blood. No one was present when the last of the blood ran in. When a nurse returned to the room, the patient was unconscious, cyanotic, having gurgling respirations and convulsing. Efforts at resuscitation failed. This death was due to massive air embolism. Another patient with placenta previa whose blood type was O, Rh positive was given 500 cc. of AB, Rh positive blood. She promptly developed oliguria and eventually died of uremia. She had very high anti-A, anti-B blood titers prior to her death.

Laboratory error in another case resulted in a sensitized Rh negative patient getting Rh positive blood. She developed shock after getting 600 cc.

of Rh positive blood, and in the treatment of the shock she got a total of 2,400 cc. of incompatible blood. Laboratory findings indicated rapid hemolysis of all of the blood administered in that no Rh positive cells could be found in blood sent to a blood bank after the transfusions. This patient expired in a matter of hours. The fourth transfusion accident occurred in a patient with hypertensive toxemia who was delivered by cesarean section. Her blood loss was treated by 1,000 cc. blood replacement plus 250 cc. of 5 per cent glucose in normal saline in a little less than one hour. About one hour later she developed acute left heart failure thought to be precipitated by the rapid blood administration. This occurred in a patient whose heart load was already increased by her hypertension. Three of the transfusion accident deaths were considered preventable.

Two suicide deaths in the puerperium were considered as "obstetric." These patients followed a similar behavior pattern showing agitation and anxiety expressed as a strong feeling of inadequacy in caring for the baby. They were regarded as normal prior to the puerperium, and the seriousness of their depression was not recognized despite unusual expressions of concern over the baby's care, both to family and physicians.

The infection deaths are accounted for by two infected criminal abortions, an undiagnosed appendicitis with rupture, and one puerperal infection. The patient with puerperal infection was hospitalized at thirty-four weeks of gestation with a small amount of vaginal bleeding and ruptured membranes. Nothing further developed at that time, and she was discharged from the hospital and not seen for ten days. At that time she was re-admitted in shock due to blood loss and was ready for delivery. Despite blood administration, which corrected the immediate shock picture, and antibiotic therapy, she followed a down-hill course and died of septicemia with peritonitis and pulmonary edema. The infection had its onset prior to the admission for delivery. The patient with appendicitis was treated for what was thought to be a threatened abortion. Neither examination nor laboratory studies were done to establish this as the cause of her pain or to rule out other causes of an acute abdomen. The diagnosis of appendicitis was not entertained until autopsy.

There were no deaths from inhalation or spinal anesthesia. However, one patient developed shock, convulsed and died after the use of one of the new local anesthetic agents. One patient was given an ampule of obstetrical pituitrin into the uterus and one ampule of pituitrin intravenously at the time of a cesarean section, whereupon she died immediately. This was a pituitrin shock under anesthesia. Obstetrical pitocin in smaller quantities would have been the drug of choice.

The "non-obstetrical" causes of death require little elucidation. Of those due to peritonitis, two were due to postoperative complications after cholecystectomy. The third in this group had peritonitis secondary to perforation of a carcinoma of the recto-sigmoid.

Summary and Conclusions

1. A brief general summary of the results of the Minnesota Maternal Mortality Study for the period from April 1, 1953, through March 31, 1954, has been presented. Certain comparisons are made between these results and those of four previous studies.

2. There were fifty-nine deaths associated with pregnancy or in the postpartum period in the 1953-1954 period among 79,362 live births, for an over-all maternal mortality rate of 0.74 per 1000 live births. When deaths due to "non-obstetrical" complications are excluded the rate is 0.42 per 1000 live births. These rates are essentially the same as for the previous two years.

3. There was an increase in the percentage of deaths considered preventable to 25.4 per cent as compared to 17.2 per cent last year. However, this is better than the 1950-1952 average of 30.6 per cent, and well ahead of the 1941-1942 figure of 73.2 per cent preventable deaths.

4. The percentage of deaths associated with pregnancy but due to "non-obstetrical" causes continues to increase. There were 44.1 per cent non-obstetrical deaths in this series as compared to 35.5 per cent for the 1950-1952 average and 17.0 per cent in the 1941-1942 study. This is due to a reduction in the "obstetrical" deaths rather than to any increase in the total number of cases.

5. Approximately the same proportion of delivered patients who died in 1953 were delivered by operative means as in the previous three years. Three, or 12.5 per cent of the operative deliveries, were considered as not being indicated.

6. The previously reported inaccuracies and in-

(Continued on Page 431)

Radioiodine Uptake in the Diagnosis of Thyroiditis

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THE classical presenting symptom of thyroiditis is thyroid tenderness. When this is present, there is seldom doubt that the patient has the disease. However, it is easy to overlook this finding if the patient does not mention it; and because of the other symptoms, the case may be mistaken for a diffuse hyperplastic goiter. Since the advent of the thyroid uptake test with radioiodine, clinicians have learned the value of this test in making the differentiation. The purpose of this report is to show how the test has been helpful in this respect.

Thyroiditis is not a common disease, and no one is afforded the opportunity of observing a great number of cases. The pathological concept generally falls into three categories: (1) the subacute form, (2) the lymphocytic type (Hashimoto's struma or struma lymphomotosa), and (3) the fibrous type (Riedel's struma or struma fibrosa). Of these three, the subacute type is the most frequent and, consequently, most important.

Thyroiditis is most prevalent in women. It runs a chronic course over many months. The thyroid is symmetrically enlarged and of firm consistency, and it is tender. Hoarseness, dysphagia, and choking are commonly mentioned. In addition, there are generalized symptoms of anxiety, palpitation, nervousness, fatigue, and weight loss. There is usually more or less tachycardia. The basal metabolic rate is often elevated, which is to be expected in a nervous, anxious individual, although this has also been explained on the basis of destruction of glandular acini with release of the contained hormone into the circulation.[2] Since these symptoms and signs are so similar to those of Grave's disease, it happens not infrequently that a thyroid gland, removed under the impression that it is toxic, is reported by the pathologist as thyroiditis. In fact, until recent years, the diagnosis was seldom made preoperatively.

During the past eighteen months the opportunity was presented of studying ten cases of thyroiditis by the I^{131} uptake method (Table I). Two of these had been followed for several months clinically as classic cases of thyroiditis. Pathological confirmation was obtained in three of the remaining eight, the others not being subjected to any surgical procedure. The normal range of I^{131} uptake by the thyroid gland in twenty-four hours is considered to be 15 to 45 per cent in our laboratory. In two of the cases (Case 3 and Case 4), the uptake was in the lower portion of the normal range, and in the other eight it was subnormal, in five being very low—consistent with myxedema. It is noteworthy that the two cases which fell in the normal range were both found on pathological examination to be Hashimoto's struma. This is in agreement with the observations of others. (See below). In general, the I^{131} uptake test does not have the diagnostic value in distinguishing Hashimoto's struma that it does in subacute thyroiditis since the uptake may range all the way from subnormal to high. The test was particularly helpful in Case 3, which was being considered clinically as a recurrent thyrotoxicosis. Of special interest also was Case 7. This patient had been tested eleven months previously because of an adenoma in the left lobe and found to have an uptake of 28 per cent. In contrast, her uptake was only 10.8 per cent shortly after the onset of thyroiditis.

In 1950, Hamilton, Kirkendall, and Barker[2] reported the uptake of I^{131} and the plasma protein bound iodine in four cases of subacute thyroiditis. They found the I^{131} uptake to be very low, and the PBI to be at high normal or elevated levels at an early stage in the disease, but both of these approached normal later in the course of the disease.

Tschumay, Beierwaltes, and Lampe[1] make the same statement, with a distinction between behavior of the I^{131} uptake early and late in the disease.

Furr and Crile[3] collected a series of sixty-two cases of struma lymphomotosa over a twelve-year

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TABLE I.

Case No.	Patient	Sex	Age	I-131 Uptake	Clinical Data	Duration	Pathology
1	L.A.	F	35	5-25-54 2.4%	Fatigue, irritability; feels cold; Soreness on swallowing. 10 lb. weight loss. Thyroid enlarged three times normal, tender. Pulse 98.	6-8 wks.	Thyroidectomy 6-24-54 Path: Chronic exudative thyroiditis.
2	H.B.	F	42	8-13-54 14.%	Had thyroidectomy in 1936. Now nervous, tremorous, weak, losing weight. Pulse, 84. Thyroid tender, nodular, slightly enlarged.	not known	
3	A.K.	F	58	1-15-54 16.9% 4-23-54 20.4%	Thyroidectomy in 1933. Palpitation, anxiety, nervousness, tightness in neck. No thyroid tenderness. Thyroid enlarged four times normal, firm. Pulse, 80.	4 mos.	Needle biopsy 5-6-54 Path: Hashimoto's struma
4	B.L.	F	44	8-9-54 22.5%	Goiter for many years, enlarging for past year. Dysphagia. Thyroid four times normal, tender. Pulse, 62.	1 mo.	Thyroidectomy Path: Hashimoto's struma
5	C.L.	F	25	8-4-54 4.%	Thyroid slightly enlarged and tender for seven months. Followed clinically as thyroiditis.	7 mos.	
6	H.S.	F	40	7-15-54 14.%	Followed clinically as thyroiditis. Thyroid tender, nodular and enlarged.	8 mos.	
7	D.W.	F	75	3-18-55 10.8%	Fatigue, dysphagia. Recent enlargement of adenoma of left lobe, probably due to hemorrhage. Pulse, 82. Eleven months earlier, I-131 uptake was 28%, when none of above symptoms were present.	2 wks.	
8	C.T.	F	34	9-14-55 0.5%	Tired, nervous; hair dry. Thyroid enlarged two times, tender, firm. Pulse, 100.	3 mos.	
9	J.Z.	F	40	11-2-55 1.2%	Anxiety, irritability, slight hoarseness and dysphagia. Thyroid enlarged, nodular, firm, tender. Pulse, 96.	4 wks.	
10	R.S.	F	40	7-13-55 1.3%	Thyroidectomy twenty years ago. Pain in neck. Dysphagia, hoarseness. Enlarged, firm, tender left lobe of thyroid. Pulse, 100.	1 mo.	

period at the Cleveland Clinic. Seventeen of these had I^{131} uptakes, all of which were normal.

Recently Skillern, Crile, et al[4] published another study of thirty-six cases of Hashimoto's struma in which they pointed out the wide range of the I^{131} uptake in this disease and concluded that it is not a good measure of thyroid deficiency in this syndrome.

Dr. Joseph M. Ryan,[5] of Saint Paul, has tested with I^{131} two cases of Hashimoto's struma, one with an uptake of 41 per cent which was confirmed surgically, and the other with a 56 per cent uptake being a clinical diagnosis.

Evans[6] followed two cases of thyroiditis with serial uptakes, one for eight months and the other for sixteen months. The initial uptakes in the first weeks of the disease were under 5 per cent, but these rose to normal levels within two to three months, following satisfactory clinical response to radiation therapy.

In our series of ten cases there is no evident relationship between the uptake and the duration of the disease, although serial uptakes were not done. The only conclusion warranted in this respect is that the low uptake reflects a poorly-functioning, diseased thyroid. With subsidence of the inflammation and return to normal function, whether this occurs in a few months or requires a year or more, the uptake can be expected to rise to the normal range again.

Although it is not intended here to discuss treatment of thyroiditis, the low I^{131} uptake suggests the rationality of administering whole thyroid extract during the course of the disease. Surgery does not seem to be indicated unless malignancy is suspected. Needle biopsy should be very useful in mitigating such suspicion. The extensive lymphocytic infiltration of Hashimoto's struma validates the rationale of such lymphocytolytic agents as radiation therapy and cortisone.

Summary

The thyroid uptake test with I^{131} is useful in substantiating the diagnosis of subacute thyroiditis since the avidity of the gland for iodine is depressed during the duration of this disease. However, in Hashimoto's struma the I^{131} uptake is variable. The test is also a useful guide in treatment since it indicates when supplemental whole thyroid should be given, along with other measures.

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Congenital Peripheral Arteriovenous Communications

Use of Femoral Artery to Heart Circulation Time in Diagnosis

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SOL SANDHAUS, B.S., and
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A CONCEPT of abnormal arteriovenous communications was first described by William Hunter[1] in 1757, however the presence of such communications as a normal physiological unit or as a congenital malformation, was not discussed until the writings of Hoyer,[2] Schumaker,[3] Grosser[4] and others in the later nineteenth and twentieth centuries. Recently, Pilulachs[5] and Cordier[6] have suggested that some instances of venous varicosities are related to the presence of abnormal congenital peripheral arteriovenous communications, and have demonstrated their findings by arteriographic means. Thus, it seems that all individuals have peripheral arteriovenous communications present in the extremities and that the variations from normal are only one of degree (Fig. 1). However, in the abnormal state, symptoms of such malformations may range from the presence of simple varicose veins, to cardiac failure.

The diagnosis of traumatic arteriovenous communications is not a difficult one. The usual signs of a bruit, localized pain, and often a pulsating mass, are due to a large blood flow via the artery to vein shunt. However, in small congenital arteriovenous shunts, this is not true. The flow via each such lesion is small, but the total potential of numerous communications may be quite large. Grant and Bland[7] have estimated the total cross sectional area of potential arteriovenous communications, as represented by the glomus bodies of the hand, to be over 1209 square cm., and even greater in the foot and leg. The flow via abnormal communications seldom represents such a tremendous transport of blood volume; however, in one instance in this series, the volume was enough to cause a marked cardiac enlargement in a six-year-old child without evidence of other cardiac disease.

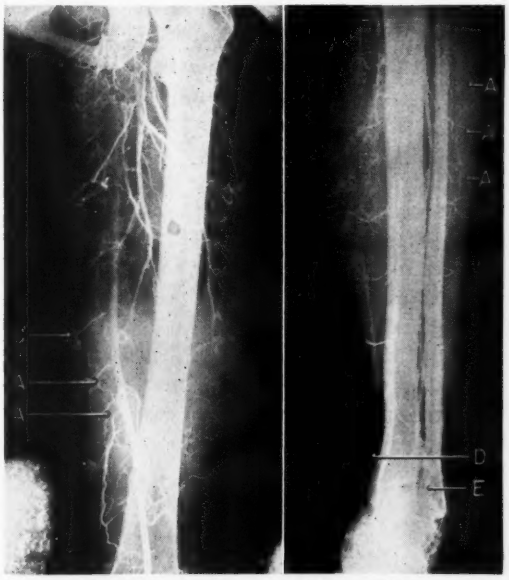

Fig. 1. Femoral arteriogram showing multiple peripheral congenital arteriovenous communications. (A) Abnormal segmentation of the main arterial stem. (C) Arteriovenous communication in the thigh. (D) Contrast material in the vein from communications around the ankle. (E) Contrast material in a vein from communications at the ankle and the foot.

Blood carbon dioxide and oxygen analyses studies may be used to give a measure of flow via these lesions. However, such determinations are expensive and necessitate the use of complicated laboratory instruments. Arteriographic demonstration of the lesions give a clear and definite idea of the communications, but necessitates hospitalization and a general anesthesia. The use of simple femoral artery to heart circula-

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TABLE I. CIRCULATION TIME. FEMORAL ARTERY TO RIGHT AURICLE APPEARANCE TIME

Normal Studies Age	Time
3 months	9.0 seconds
20 years	20.0 seconds
58 years	25.2 seconds
Adult Mean Time (12 studies)	21.0 seconds
Arteriovenous Communications	
6 years	1.0 seconds
19 years	8.5 seconds
21 years	7.8 seconds
32 years	8.8 seconds
46 years	6.6 seconds

tion time using a radioactive tracer element, I^{131}, presents a simple, safe and accurate means of diagnosis and study of these lesions.

Method

Thirty to 40 microcuries of iodine^{131} labeled human serum albumen in sterile saline, are injected rapidly into the femoral artery under local anesthesia, using a No. 20 hypodermic needle. A lead collimated scintillation counter, located over the right auricle, 1 to 3 inches above the xiphoid and to the right of the sternum, detects the appearance of the radioactivity when it arrives in this area. The radioactivity level detected by the counter is recorded continuously by a ratemeter and graphic recorder. The time from the injection to the appearance of radioactivity at the right auricle is called the femoral artery to heart appearance time.

During the procedure, the patient is placed in a constant temperature room at 20° C., in a condition approaching a basal state of activity. Under these conditions, the normal arteriovenous communications, the glomus units of heat regulatory control, should be closed.

The equipment used other than that necessary for any intra-arterial injections, include:

1. Tracerlab P-20 scintillation detector and mobile support stand.
2. Tracerlab SC-34 precision ratemeter.
3. Esterline-Angus (0-1 MA.) recorder.

Most of this equipment is found in the usual biomedical laboratory equipped for work with radioactive elements. The dosage of radioactivity administered is very small and may be repeated if necessary.

Results

Femoral artery to heart circulation times have been studied in a group of patients without demonstrable vascular disease, in ages from three months to fifty-eight years, and in both male and female patients (Table I). The adult range of normal lay between 20 to 25.2 seconds, while as expected, in a small child the time was less, being 9.0 seconds in an infant of three months of age. The adult mean time was 21 seconds.

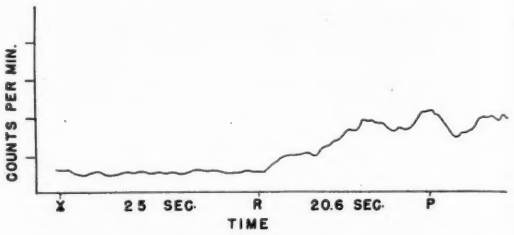

Fig. 2. Graphic representation of radioactivity at the right auricle in a normal study. (X-time of injection into the femoral artery) (R-appearance of radioactivity at the right auricle).

Five patients with abnormal arteriovenous communications were also studied. These lesions were later demonstrated by arteriographic studies in all, and a surgical exploration in an attempt of correction, in three of these patients. The range of the circulation time in these instances was 1.0 seconds to 8.8 seconds. In none of this group in which these lesions were suspected, did the circulation time even approach that of a three-month-old normal infant (Figs. 2, 3 and 4).

Discussion

The rate of blood flow from the femoral artery to its return to the right heart is inversely proportional to the degree of peripheral resistance in the arterial bed, and directly proportional to the "vis a tergo"* in the venous system. Thus, we may expect, that in the presence of arteriovenous malformations which do not respond to normal physiological control, that the peripheral resistance of this area is lessened, and further, that there is a transfer of kinetic energy to the venous system. As such, the rate of blood flow via this pathway is increased. The measure of this flow rate was attempted by Pilulachs,[5] using diodrast and its identification by taste sensation at the tongue. The results in this experiment were crude due to the failure and delay of taste sensation. Secondly, the contrast agent was a hyperbaric solution and of a high viscosity, which did not mix well with flowing blood. The use of iodinated human serum albumen in this experiment has alleviated these

*"Force from behind."

difficulties. Further, the rapid formation of a flat level of radiocativity at the heart, following the injection of the test agent, indicates that a very rapid diffusion of the "tagged" albumen throughout the system takes place.

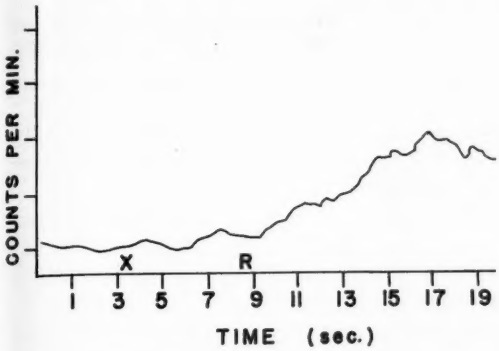

Fig. 3. Graphic representation of radioactivity at the right auricle in an abnormal study (arteriovenous communications). (X-time of injection into the femoral artery.) (R-time of appearance at the right auricle.) (Circulation time of 8:5 seconds.)

The correlation of the radioactivity curve at the right auricle, to the degree of involvement of an extremity with arteriovenous malformations, has been attempted. To date, we have not been able to correlate the type or configuration of this curve to be of any significance. We do not see the "bicamel" type curve as described with the use of Evans Blue dye dilutions techniques,[8] presumedly because the pick-up of radioactivity by the detecting tube is not shielded against the return of radioactivity following its transit through the lung and into the left heart. It is, significant however, that in the one patient in which we have demonstrated massive numbers of malformations, the appearance time at the right auricle was the shortest of the series, one second.

Summary

A simple diagnostic test in screening patients for the presence of small congenital arteriovenous communications of an extremity is presented, with the use of a femoral artery to heart circulation time using a radioactive tracer element, I^{131}. This is a simple and safe procedure, and one that may be carried out in any laboratory equipped for the use of radioactivity. It may be suggested that this same technique could be utilized to measure other blood transit times in evaluating the patency of porto-caval or spleno-renal shunts by injecting into a hemorrhoidal vein and measuring the time of arrival of radioactivity at the heart, and the problem of venous drainage of the extremity in the "post-phlebitic" syndrome.

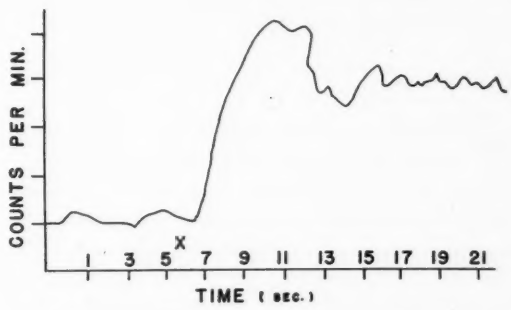

Fig. 4. Graphic representation of radioactivity at the right auricle in an abnormal study, arteriovenous communications. (X-time of injection into the femoral artery.) (Circulation time of 1.0 second.)

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TODAY'S HEALTH PROBLEMS

Today it is generally accepted that an educated and enlightened group of citizens can and do best solve their own health problems, those of their families and their communities. Moreover, the nature of today's health problems—chronic disease, mental health, and accident prevention—demands that in addition to technical skills all health workers must be capable of utilizing educational and community organization procedures. We must not be guilty of telling people what to do, but we must help them become the kind of people who will know what to do.—L. E. Burney, M.D., *California Medicine*, January, 1956.

Accidents of Children

A Study of Accidents of Injured Children Seen at the Mayo Clinic in 1954

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Rochester, Minnesota

ACCIDENTS are responsible for more deaths in infants and children between the ages of one and fourteen years than the four most fatal diseases of childhood combined, namely, pneumonia, congenital defects, cancer and tuberculosis.[1] In 1952, although Minnesota had its worst epidemic of poliomyelitis, accidents accounted for the deaths of two and a half times as many children as polio did.[2] Despite the fact that nearly all childhood accidents are preventable, the death rate from accidents among children is being reduced only about a third as fast as the death rate from disease.[3]

Before the accident problem can be solved, it must be defined.[4-6] It is best defined by statistics: From 11,000 to 13,000 children are killed by accidents in the United States each year; that is, one-third the total number of children who die each year do so as the result of accidents. From 40,000 to 50,000 is an estimate of the number of children permanently injured each year in the United States by accidents.[7] Approximately 1,000,000 children receive medical care each year because of accidents.

The prevention of accidents to children then is a major and still unsolved problem. Its solution should be the concern not only of the pediatrician but of the general practitioner and all other physicians and surgeons who deal in some way with the care of children who are injured accidentally.

This study was undertaken to ascertain the types and causes of acute accidents to children in a small urban and rural community. During the year, 1954, Rochester and Olmsted County had only two hospitals, both affiliated with the Mayo Clinic. A unique opportunity, therefore, was available for such an inquiry.

The study was limited to children less than fifteen years old who were seen either as outpatients in the emergency rooms or as inpatients in one of the two hospitals. Almost all of the children were from Olmsted County. Children seen at home by physicians or treated by the parents without being seen by a physician were not included; nor were those who had sudden fatal injuries, such as drowning or being killed outright in an automobile accident.

This study therefore, encompasses the usual types of accidents seen in this kind of community. It is not a compilation of the extremely unusual or rare type of accident.

Method of Study

Data concerning the accidents and injuries were obtained from the records on file in the Mayo Clinic. The accidents were of four major types: (1) home, (2) public, (3) motor vehicle and (4) occupational (or school). Observation of this classification immediately shows some overlapping as the accidents in the public domain naturally would include many motor vehicle accidents and others might be in the home area. For our purposes we defined the groups as follows:

Included as home accidents were any accidents which happened in or around the home proper. Public accidents consisted of those that occurred away from the home, on the street, sidewalk or any other place of public recreation, amusement or activity. Motor vehicle accidents consisted of those in which the child was injured as a passenger in an automobile, by collision of the automobile, sudden stopping, or falling from the moving vehicle. Accidents in which the child, as a pedestrian, was injured by a motor vehicle, were classed as public accidents. Accidents resulting

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from a closing car door, the majority of which occurred in the home area proper, were included under the major classification of home accidents, or if the injury occurred away from home as public accidents. Occupational or school accidents were those which occurred on the school grounds or in the school.

The principal cause of each accident was determined. Causes were found to be falls, unsupervised activity, supervised activity, machinery, foreign body, motor vehicles, poisoning, burns, firearms, suffocation and some miscellaneous ones. In a sense unsupervised activities would cover many of the other causes. However, they have been listed separately because of their importance. In this list of principal causes of accidents, accidents involving motor vehicles whether the child was a pedestrian or passenger were considered to be due to motor vehicles. The only exceptions were the accidents due to closing of car doors and windows. Because of the specific nature of the injury and the rather large number of such accidents, these accidents were classed as being caused by doors and windows.

It became apparent during the study that the terms "falls and miscellaneous" could more appropriately be considered as subdivisions in the groups of accidents due to (1) unsupervised and (2) supervised activities or play; or as (3) those associated with animals, foreign objects and door and window injuries.

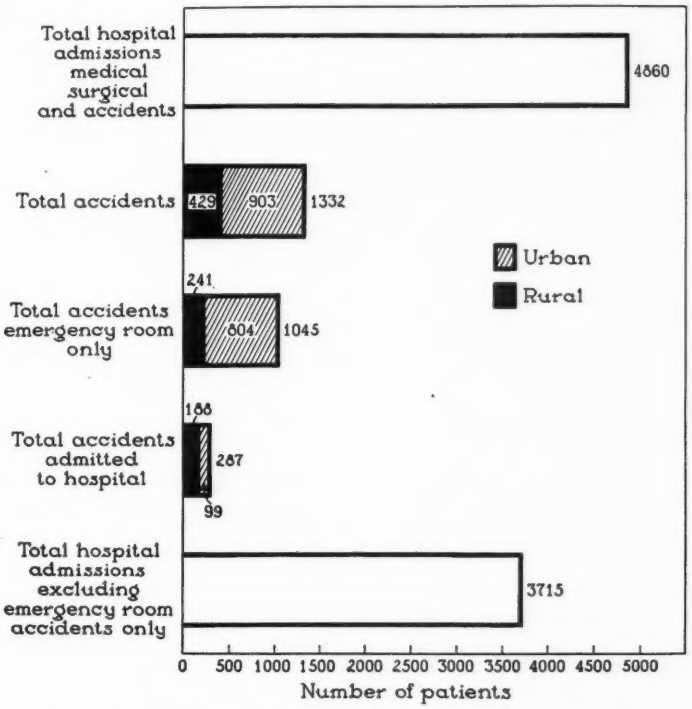

Fig. 1. Comparison of hospital admissions and accidents of children in Rochester in 1954.

Since accidents due to foreign bodies are more closely associated with children than adults, this cause of accidents was subdivided as to type: (1) aspiration or ingestion, (2) penetrating or cutting and (3) self-introduced.

Number of Children Injured

Of the 4,860 children seen in 1954 in the emergency rooms or in the hospitals of Rochester, 1,332 had had accidents (Fig. 1). Thus 27.4 per cent of the patients seen on the pediatric services under these circumstances were seen because they had injuries. Of the injured patients 903 were from the urban area and 429 from the rural area. One thousand forty-five of the children injured needed emergency room treatment only; 804 of these came from the urban area and 241 from the

rural area. The remaining 287 children who were injured were hospitalized; ninety-nine of these were from the urban area and 188 from the rural areas.

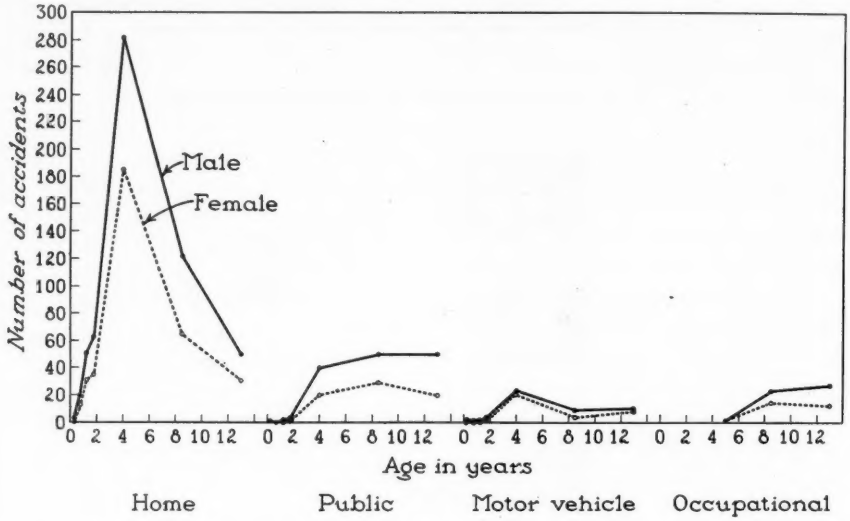

Fig. 2. Major classification of accidents of children according to sex and age.

The number of admissions to the hospitals' pediatric services for all causes in 1954 was a total of 3,815. Hence, approximately 7 per cent of our hospital admissions of children for 1954 were for injuries sustained in accidents.

Type of Accidents

The type of accident and the sex and age of the patients are indicated in Figure 2. In the entire group, there were 837 males and 495 females injured, a ratio of one female to 1.7 males.

The greatest number of accidents to our patients occurred in the home and involved children from two to five most frequently and those from six to ten years of age next. These home accidents numbered 953 (71.5 per cent of the total).

Two hundred seventeen accidents (16.3 per cent) occurred in public places. Children from six to fourteen years old were involved most frequently with about an equal number for ages six to ten and eleven to fourteen. Younger children had somewhat fewer accidents away from home.

The greatest number of children injured as passengers in or by motor vehicles was in the age group of two to five years. The total number of children having motor vehicle injuries as passengers was eighty-four (6.3 per cent).

Seventy-eight children (5.9 per cent) were injured in occupational types of accidents. The two occupational accidents in the age group of two to five years were those of two five-year-old children who were in kindergarten.

Urban Versus Rural Accidents

Nine hundred and three children were injured in the city and 429 in the rural areas. Figure 3 relates the type of accident to its occurrence in the city or in a rural area. The ratio of urban accidents to rural accidents is 2:1 in this series of cases.

The 1950 United States[7] census indicated that a total of 12,803 children through the age of fourteen years were living in Rochester and Olmsted County areas. Of this total, 6,682 children were in urban Rochester and 6,181 were in the rural areas of Olmsted County. The total figures are undoubtedly somewhat higher for 1954, but the ratio of urban children to rural children is probably the same. From use of the 1950 census figures and the 1954 hospital-accident figures, the incidence of urban accidents was one accident for every seven children, and the rural incidence was one for every fourteen children.

The unusually high incidence of urban accidents

in this series may be accounted for in part by the ease with which children of the community can be brought to the hospitals for all types of emergency treatment.

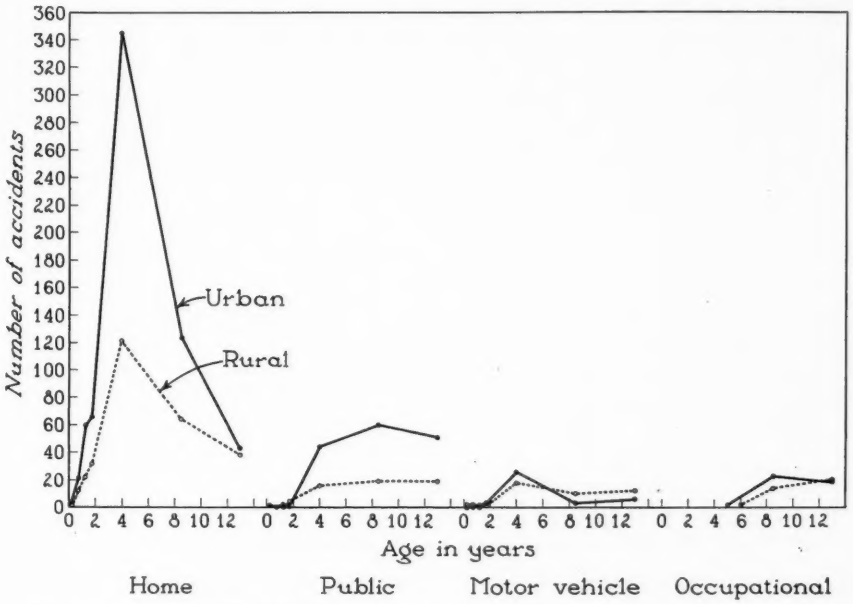

Fig. 3. Major classification of accidents of children according to locality, urban and rural, and to age.

A critical analysis of the figures on the principal cause of the accidents will show that more rural than urban children were injured by motor vehicles (both as pedestrians and passengers) and machinery. Further evidence of the seriousness of rural accidents will be discussed later in relation to the residual resulting from the accidents.

Principal Causes

The principal causes of the accidents in relation to age and sex of the children and site of the accident, whether urban or rural, are listed in Tables I and II. A few of the principal causes were subdivided to show the type of accident more clearly. Accidents of similar nature were classed under several principal causes. For example, injuries resulting from falls were listed under unsupervised activity and supervised activity, whichever category was correct. Falls accounted for a combined total of 487 accidents, both urban and rural, or 36.6 per cent of all accidents.

Unsupervised outdoor activities were the principal cause of accidents; the total was 293 accidents (22 per cent). The next largest number of accidents occurred during unsupervised indoor activity, including falls from the porch or windows and other injuries, accounting for a total of 291 (21 per cent). Foreign-body accidents caused 201 injuries (15 per cent). These three principal causes together accounted for almost three-fifths of the accidents; that is 58.9 per cent. The remainder of the causes of accidents and the total number of cases encountered are: motor vehicle (pedestrian and passenger exclusive of door and window injury), 114 cases; supervised activities, ninety cases; poisoning, eighty-eight cases; machinery, fifty-nine cases; burns, forty-eight cases; firearms, four cases; suffocation, three cases; and miscellaneous, 141 cases, of which thirty-six cases were due to foreign objects such as rocks and stones, forty-six cases to animal type of accidents such as bites, scratches or falls, and fifty-nine cases to door and window injuries.

Frequency of Injury in Certain Age Groups.—From both Tables I and II it may be seen that children between the ages of two and five years are injured accidentally much more frequently than those of other age groups. There was a total of 575 children from two to five years old (43

TABLE I. PRINCIPAL CAUSES OF 903 ACCIDENTS OF URBAN CHILDREN

Principal Cause	Total		Age and Sex													
			Less than 6 mo.		6-11 mo.		12-17 mo.		18-23 mo.		2-5 yr.		6-10 yr.		11-14 yr.	
	Number	Per Cent	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Unsupervised activities (out of doors)	213	23.6					4	2	4	3	60	33	53	28	21	5
Falls	150	16.6					4	1	2	2	38	25	36	19	19	4
Miscellaneous	63	7.0						1	2	1	22	8	17	9	2	1
Unsupervised activities (in or about the house)*	229	25.3		2	7	6	16	6	23	12	67	48	15	19	3	5
Falls	197	21.8		2	6	6	15	5	21	10	57	42	12	16	1	4
Miscellaneous	32	3.5			1		1	1	2	2	10	6	3	3	2	1
Foreign bodies	133	14.7	1		2		2	3	7	2	40	24	20	8	14	10
Penetrating	101	11.2					1	3	5	1	32	13	16	6	14	10
Aspiration-ingestion	23	2.5	1		2		1		2		4	8	3	2		
Self-introduced	9	1.0								1	4	3	1			
Motor vehicle†	54	6.0					1		2	1	17	12	8	2	8	3
Supervised activities	66	7.3		1							4	2	18	11	20	10
Falls	21	2.3									2	1	5	7	4	2
Miscellaneous	45	5.0		1							2	1	13	4	16	8
Poisoning	58	6.4			2	1	9	3	6	2	19	15				1
Machinery	23	2.6									9	6	1	1	2	4
Burns	26	2.9					5	4			5	6	4		1	1
Firearms	2	0.2											1		1	
Suffocation	2	0.2	1								1					
Miscellaneous	97	10.8			2		4	2	6	1	28	24	9	12	5	4
Door and window	45	5.0			1		4	1	3	1	16	10	2	3	1	3
Animal	25	2.8						1	2		3	10	2	5	1	1
Foreign object	27	3.0			1				1		9	4	5	4	3	
Total	903	100.0	2	3	13	7	41	20	48	21	250	170	129	81	75	43
Grand Total			5		20		61		69		420		210		118	

*Indoor activities, including falls from the porch.
†Includes all types whether public, that is, pedestrian or passenger. Excludes doors and windows.

per cent) of the total. The 1950 United States census statistics for Rochester and Olmsted County list approximately 4,034 children between the ages of two and five years in this area, about 32 per cent of the total number less than fifteen years of age. Though the 1954 population figures are undoubtedly increased, the age-group proportions and percentage of children are probably the same.

These figures then tend to substantiate the findings of others[4,6] that children between the ages of two and five years are injured more frequently by accidents than children in any other age group.

Types of Injury

The types of injuries are grouped in order of frequency in Table III. Lacerations accounted for 38.2 per cent of the total injuries.

A few of the objects responsible for penetrating and cutting injuries in order of frequency are (1) metal objects such as cans, coat hangers, and metal toys, (2) nails, (3) glass, (4) wood, (5) utensils, both kitchen and garden, (6) arrows or darts and (7) a miscellaneous group.

The ingestion or aspiration of foreign bodies constitutes a small (forty-eight cases) but significant number of accidents. The kinds of objects in the order of frequency were (1) coins, (2) foodstuffs such as peanuts, fishbones, popcorn, meat, carrots, orange peel and egg shells, (3) metallic objects as bolts, screws and tacks, (4) pins and buttons and (5) glass beads and marbles. The most unique object was a lead cow-bell clapper.

Self-introduced foreign bodies were found most commonly in nasal passages, occasionally in the external auditory canal and rarely in the vagina. The various types of objects in order of frequency were pins, buttons, glass beads, paper, gum and crayons.

Poisoning is an important cause of accidents. There were eighty-eight children in our group who were injured in this way. A partial list of the different types of poisons includes in order of frequency: aspirin, turpentine, fuel oils, arsenic, dilantin, lye or bleach, liquid soaps, nicotine, insecticides and single instances of poisoning by a great variety of medicines and other substances to which children have access in homes.

Here as elsewhere,[8] aspirin is an important and dangerous cause of poisoning. The same may be said of turpentine and fuel oil both of which are often easily accessible in soft-drink and other lightly stoppered or unstoppered bottles. The easy accessibility of these and other things (the fault of an adult) is responsible for the poisonings.

TABLE II. PRINCIPAL CAUSES OF 429 ACCIDENTS OF RURAL CHILDREN

Principal Cause	Total		Age and Sex													
			Less than 6 mo.		6-11 mo.		12-17 mo.		18-23 mo.		2-5 yr.		6-10 yr.		11-14 yr.	
	Number	Per Cent	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Unsupervised activities (out of doors)	80	18.6				1	1	1	1	1	24	4	23	9	10	5
Falls	55	12.8				1	1		1		12	3	20	8	5	4
Miscellaneous	25	5.8						1		1	12	1	3	1	5	1
Unsupervised activities (in or about the house)*	62	14.4			2	1	2	3	5	2	22	13	5	2	5	
Falls	52	12.1			2	1	2	3	4	2	19	10	5	2	2	
Miscellaneous	10	2.3							1		3	3			3	
Foreign bodies	68	15.9			2	2		1	7	4	14	9	11	7	5	6
Penetrating	35	8.2							2	2	3	6	9	4	4	5
Aspiration-ingestion	25	5.8			2	2		1	5	1	8	1	1	2	1	1
Self-introduced	8	1.9								1	3	2	1	1		
Motor vehicle	60	14.0	2		1				3	2	12	12	10	4	7	7
Supervised activities	24	5.6											5	4	10	5
Falls	12	2.8											4	3	2	3
Miscellaneous	12	2.8											1	1	8	2
Poisoning	30	7.0					3	3	5	2	9	6		1	1	
Machinery	36	8.4					2	1		1	7	7	8	2	8	
Burns	22	5.1		1	3	1	2	2		3	3	2	3		2	
Firearms	2	0.5													1	1
Suffocation	1	0.2	1													
Miscellaneous	44	10.3	1				2			1	6	5	9	3	13	4
Door and window	14	3.3	1				2			1	4	1	2	2	1	
Animal	21	4.9									1	3	2	1	10	4
Foreign object	9	2.1									1	1	5		2	
Total	429	100.0	4	1	8	5	12	11	21	16	97	58	74	32	62	28
Grand Total			5		13		23		37		155		106		90	

*Indoor activities, including falls from the porch.

TABLE III. TYPES OF INJURIES IN 1,332 CHILDREN

Types of Injury	Total	Urban				Rural			
		No.	%	M	F	No.	%	M	F
Lacerations	540	399	28.2	280	119	141	10.0	94	47
Fractures and dislocations	220	111	7.9	60	51	109	7.7	70	39
Contusions	197	134	9.5	75	59	63	4.5	36	27
Head injury	117	71	5.0	41	30	46	3.3	31	15
Abrasions	112	77	5.4	41	36	35	2.5	21	14
Poisoning	88	58	4.1	36	22	30	2.1	18	12
Burns	48	26	1.8	15	11	22	1.5	13	9
Aspiration or ingestion	48	23	1.6	13	10	25	1.8	17	8
Sprains	24	20	1.4	13	7	4	0.3	3	1
Crush injury	14	3	0.2	3	—	11	0.8	10	1
Gunshot	4	2	0.1	2	—	2	0.1	1	1
Suffocation	3	2	0.1	2	—	1	0.1	1	—
Total	1,445	926*	65.3	581	345	489*	34.7	315	174

*Some children had more than one type of injury in a single accident.

Permanent Disabilities

Of the fourteen urban children in the total of 903 who suffered permanent disability, two were less than two years of age; the youngest was eleven months of age; four were between two and five years, three were six to ten years and five were eleven to fourteen years. Of the thirty-nine rural children who incurred disabilities, six were less than two years of age; the youngest was ten months; nine were two to five years old, nine six to ten years and fifteen were eleven to fourteen years old.

The types of accidents which gave rise to the permanent disabilities of the urban children were in order of frequency: unsupervised activities, foreign bodies, and firearms, three cases each; burns, two cases; and suffocation, animal and door and window injuries, one case each. Some of the residua of these fourteen urban children were peripheral nerve palsies with associated loss of function, traumatic cataracts, extensive scarring with contraction deformities, amputations and a single case of decerebrate rigidity (the result of accidental hanging).

The types of accidents of rural children that resulted in permanent disability are decidedly different from the foregoing and include ten due to machinery, eight to burns, seven to foreign bodies, six to motor vehicles, four to animal injuries, three to unsupervised activity and one to poisoning.

The permanent damage resulting from the

accidents of the rural children include in order of frequency: (1) peripheral nerve palsies with loss of function; (2) extensive scars with contraction deformities; (3) loss of function of a whole or part of an extremity without nerve injury; (4) amputations, varying from loss of a single phalanx to loss of an entire leg; (5) severe keloids; (6) strictures (both esophageal and urethral); (7) traumatic cataracts, and (8) total enucleations of an eye.

There was one case of permanent disability for every sixty-five urban accidents, and one for every eleven rural accidents. The over-all ratio was one case of permanent disability for every twenty-five accidents.

Whereas we encountered fewer accidents among children from our rural areas, those who were seen were more severely injured and more of them had permanent disabilities than did those from the urban area.

TABLE IV. ACCIDENTS OF CHILDREN: HOSPITAL STAY AND LENGTH OF DISABILITY

	Urban			Rural		
		Days			Days	
	Patients	Hospitalized	Of Disability	Patients	Hospitalized	Of Disability
Unsupervised activities (in or about house)*	16	81	436	19	91	236
Unsupervised activities (out of doors)	24	121	906	22	210	1,599
Supervised activities	6	35	120	9	48	348
Motor vehicle	9	58	112	31	660	2,510
Foreign bodies	14	91	501	29	170	240
Aspiration		48			80	
Penetrating		43	501		90	240
Poisoning	12	31	—	14	78	300
Burns	7	205	459	14	774	1,776
Firearms	2	18	24	1	33	90
Suffocation	1	15	—	1	2	—
Miscellaneous	8	100	199	21	237	1,865
Foreign objects		76			30	659
Animal		17			201	1,192
Door and window		7			6	14
Machinery	—	—	—	27	347	2,300
Total	99	755	2,757	188	2,650	11,264
Average days per patient		7.6	27.8		14.1	59.9

*Indoor activities, including falls from porch.

Fatal Accidents

Seven children died as a result of injuries: five were males, and two, females. Two were urban and five were rural.

Three of these, aged three days, eleven months, and seven years, respectively, died as a result of a foreign-body type of injury. The specific type in two cases was aspiration of formula and egg shells, and in one case, penetration of a wooden stick which pierced the child under its eye and entered the brain.

Two children, aged twenty-two months and six years, were injured fatally in motor vehicle accidents. In each instance the child was a pedestrian and had been struck by a motor vehicle.

One child, aged eight years, was injured fatally when she fell from a second story window at her home. A twelve-year-old boy was injured fatally when his chest was crushed by a grain wagon.

These seven fatal accidents represent an over-all ratio of one death for every 190 accidents. The urban incidence rate was one death for every 450 accidents, and the rural fatality ratio was one death for every eighty-six accidents.

Duration of Stay in Hospital and Disability

The duration of the stay in the hospital and length of disability is given in relation to the kind of accident and to whether it occurred in the urban or rural area in Table IV.

The incidence rate of hospital admissions for urban children was one for every 9.1 accidents; for rural children one for every 2.3 accidents.

The ninety-nine urban patients spent a total of 755 days in the hospitals or an average of 7.6 days per patient. The total number of days of disability of the children injured in the urban area was 2,757 or an average of 27.8 days per patient.

The 188 patients injured in rural areas spent a total of 2,650 days in the hospital, an average of 14.1 days per patient. The total number of days of disability of these rural children was 11,264, an average of 59.9 days per patient.

In 1954 about twice as many children accidentally injured in rural areas as in urban areas were hospitalized. The average duration of hospitalization was more than twice as long for the children

from the rural areas. Two types of accidents contributed chiefly to the more prolonged hospitalization of children injured in rural areas: burns and motor vehicles. Children whose burns occurred in rural areas were hospitalized on an average of twice as long (55.2 days as compared to 29.3 days) as those burned in the city. This indicates more severe burns among the rural children.

Three types of accidents contributed chiefly to the prolonged period of disability suffered by children injured in rural areas: burns, motor vehicles and machinery. We encountered no machinery injuries among urban children sufficiently severe to warrant hospitalization. The average hospital stay of 12.9 days and disability of 85.2 days among those injured by machinery on the farm should emphasize the increased hazard of living on farms since they have become mechanized. It behooves farm parents to see that the farm machinery is carefully operated and that tractors and other machines are not entrusted to children who are not capable of operating them.

Comment

It is generally agreed that most accidents among children are preventable. It can be stated also that adults are responsible for an extremely large number of the accidents that befall children.

To show these facts more clearly our study revealed that 71.5 per cent of the total number of accidents were home accidents and that children in the preschool age group, that is, two to five years, were injured about twice as frequently as were children of any other age. For at least three-fourths of the total accidents the parents could be held directly or indirectly responsible.

It is apparent that prevention of accidents must include not only protection of the child but also education of the child and of all adults whose attitude is that, "Well, accidents are bound to happen, so what." Unless the family physician or pediatrician provides the education and supports a co-ordinated state-wide educational program, many families will not be given any "safety vaccine."[9] This term has been coined to stress the necessity of adequate safety education. Its omission is as grievous as failure to administer protection against smallpox, diphtheria, tetanus and whooping cough.[10]

We as physicians and surgeons must educate ourselves to the problems of preventing accidents in children. We must emphasize the importance of accident prevention to our medical students, interns, residents, patients and parents of patients.

Summary

In 1954, 1,332 infants and children who had experienced acute accidents were seen at or admitted to the hospitals affiliated with the Mayo Clinic. Of this total, 903 came from the urban area and 429 were from the rural area; a ratio of 2:1. The ratio of accident to nonaccident hospital visits was 1:3.6 and that for hospital admission was 1:13.3.

The largest number of accidents were in the major classification of home accidents.

Two hundred seventy-eight patients (188 from rural and ninety from urban areas) were admitted to the hospitals because of accidents. The average number of days in the hospital per patient was 14.1 and 7.6, respectively, for rural and urban children. The average number of days of disability was 27.8 for patients from urban areas and 59.9 for those from rural areas.

Fifty-three children had permanent disability, one for each twenty-five accidents; seven fatalities occurred, one for each 190 accidents.

These facts emphasize the need for a better and more co-ordinated program for accident prevention in Minnesota. In order to promote an effective program doctors must develop an interest in and desire to eradicate accidents as a killer and disabler of children.

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Case Presentation

Transitional Hydatidiform Mole with Fulminating Pre-eclampsia and a Macerated Fetus

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Minneapolis, Minnesota

THIS case report, prompted by a somewhat similar one by Berry, Swerdlow and Edwards,[2] concerns a patient who developed a severe fulminating pre-eclampsia at twenty-one weeks gestation and was found to have a well-formed fetus accompanied by a transitional hydatidiform mole.

Typical hydatidiform moles are rather uncommon. Chesley, Cosgrove and Preece[4] found fifty-seven during a period when there were 75,000 deliveries, or an incidence of 1:1,321. Hertig and Edmonds[7] gave the incidence as 1:2,062 (term pregnancies). The same authors[7] quote Boivin as stating the incidence of moles in the second trimester of pregnancy is 1:20,000 pregnancies. The term "transitional hydatidiform mole" was first used by Hertig and Edmonds[7] in the article "Genesis of Hydatidiform Mole" (1940). However, in this same article they credited Gierse and Storch with suggesting that there existed some intermediate (or transitional stages) between the hydatid changes seen in the early abortus and the full-blown variety seen in the typical mole.

Hertig and Edmonds[7] found that 50 per cent of the early abortuses were pathologic ova, in which the embryo was either absent or imperfectly developed. There was some hydatidiform degeneration of the chorionic villi in these pathologic ova. They found that 80 per cent of the classic moles consist only of masses of typical hydatidiform villi. Occasionally there may be an empty chorion cavity or an amnion with or without a tiny nodular embryonic mass. In between the two extremes (of the early abortus and the classic mole) there lies the transitional type. The average age of the pregnancy in these cases was found to be 16.6 weeks (menstrual age). Hertig and Edmonds[7] found fifteen of the transitional type in their series for an incidence of 1.44 per cent. Of the fifteen, four had small macerated fetuses present; in the other eleven there was only an empty chorionic sac or an amnion cavity with a tiny nodular embryo. The chorionic villi ranged from normal villi, to large ones with vessels present, on to true hydropic villi without vessels. Usually there were enough grossly cystic ones to allow a diagnosis of a hydatidiform mole.

From Maternity and Mt. Sinai Hospitals.

Case Report

Mrs. C. G. was a twenty-eight-year-old woman, para four. All her previous pregnancies had terminated in normal spontaneous deliveries of normal infants which weighed seven and one-half to eight pounds. She had no history of hypertension or nephritis. She was first seen by me May 2, 1953, with a history of her last period beginning on February 15, giving her a due date of November 22, 1953. She was Rh positive, her blood pressure was 120/80, urinalysis was negative and physical examination was negative except findings of a normal early pregnancy (ten weeks size). Her next visit was on June 5, when no abnormalities were found. On her visit June 26, she stated she had had some vaginal bleeding of slight amount on one day since her previous visit. Blood pressure and urine were negative. About July 11, the patient called on the phone to say that her legs were swelling considerably; she was put on a low salt diet and asked to come in for a check-up in a few days if she were not feeling better. On July 14, she came in complaining of swelling, spots before the eyes, and not feeling well generally. She was found to have gained 18½ pounds in five weeks, most of it since her last visit two weeks previously. Her blood pressure at this time was 180/120, there was a 3-plus albuminuria, the fundus of the uterus was about mid-way between the umbilicus and the xiphoid process, no fetal heart sounds were heard, and there was a 3-plus pitting edema of the pre-tibial-area.

The patient was hospitalized immediately and placed on conservative toxemia therapy. This consisted of bed rest, low salt diet and sedation with morphine, magnesium sulfate phenobarbital and paraldehyde. Physical examination showed some minimal localized spasm of arterioles of the eyegrounds, and some left costo-vertebral tenderness, besides the findings noted previously. Urine showed 1-plus albuminuria and many clumps of white blood cells; the hemoglobin was 10 grams per cent. Impressions at this time were: (1) early fulminating pre-eclampsia, (2) the same plus a

hydatidiform mole, (3) acute nephritis, or (4) acute yellow atrophy. Additional laboratory reports were: Uric acid 4.3 mg. per cent, blood urea nitrogen 22.3 mg. per cent and an icteric index of 8. Since the patient had tenderness over the left kidney and many pus cells in the urine, she was placed on penicillin and streptomycin.

Fig. 1. Gross specimen. Slightly macerated 8-inch long fetus. Large placenta with areas of grape-like vesicles.

An x-ray examination of the abdomen was ordered because the uterus was a month and a half to two months larger than it should have been. There was reported no definite fetal structures although there were some shadows which might represent a small fetal thoracic cage. A quantitative test for chorionic gonadotropin was done and revealed that there was more than 200,000 mouse units per liter (80,000 m.u. being the upper limit in normal pregnancy).

Since definitive treatment of the toxemia seemed urgent (no improvement after thirty-six hours), and since it was felt that there was a hydatidiform mole present either with a dead fetus or no fetus, it was decided to interrupt the pregnancy. The blood pressure the day after admission was still 175/120, her intake was 2,100 cc. with an output of only 950 cc. Because the uterus was so large an abdominal hysterotomy was done on July 16, using N_2O-O_2, pentothal, and curare anesthesia. On opening the uterus through a classical type incision it was found to be about term size and quite soft. The uterus was almost completely filled by a very large and edematous placenta which was covered by many grape-like cystic villi. In one portion of this placenta was a sac which contained a well-developed macerated fetus which measured 20 centimeters (8 inches) from crown to heel. The entire specimen is seen in Figure 1. The cystic structures are shown grossly and microscopically in Figures 2 and 3. The cystic villi ranged in size from several millimeters to a centimeter or more in diameter. The entire placenta was covered with areas of many cystic villi and in between these areas the tissue was merely edematous. The placenta measured about 10 by 20 inches by 1 to 2 inches in thickness.

The uterine and abdominal wall incisions were repaired in routine fashion and the patient given two pints of blood. The patient was afebrile postoperatively with antibiotics. The blood pressure was down to 140/90 by the morning after operation and remained at that level. On the day of surgery her intake was 2100 cc. with an output of 350 cc. On the first postoperative day the output was equal to the intake and for the next three days it exceeded the intake. She was discharged in good condition on the seventh postoperative day.

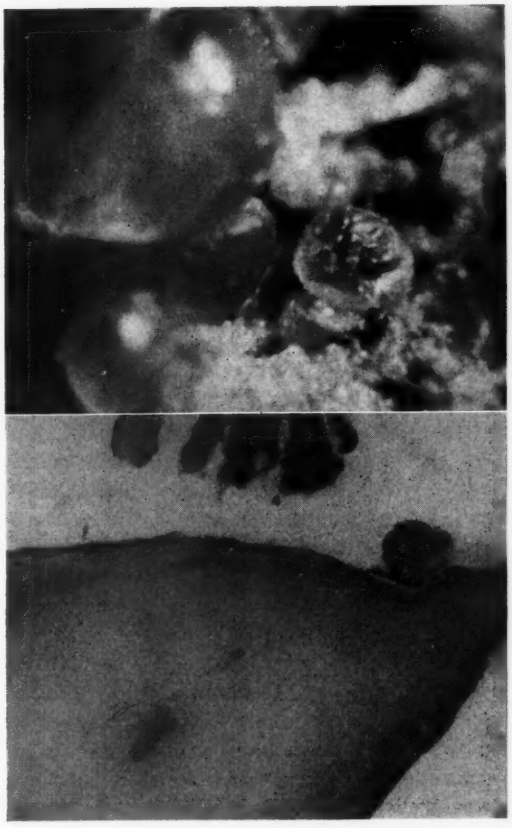

Fig. 2. *(above)* Gross specimen. Close-up view of cystic villi, three times actual size.

Fig. 3. *(below)* Microscopic section of a typical hydropic villus, magnification X 600.

Follow-up.—On August 5, the blood pressure was 120/80, the urine was negative and a pregnancy test was reported negative. She was seen at monthly intervals for about nine months with negative findings reported. The patient did not come in for a check-up from May, 1954, until September, 1954. In mid-September when she did come in she said her last period was July 21, 1954. On examination it was found she was about two months pregnant. She was checked at frequent intervals and proved to have a completely normal pregnancy. The course of this preg-

nancy was without signs of toxemia or bleeding, et cetera. A few weeks before term it was decided to deliver her by repeat cesarean section since her previous operation had been a classical type. This was done on April 15, 1954, at Mt. Sinai Hospital, Minneapolis, Minnesota. At operation the previous uterine incision was attenuated to such a degree that it was paper-thin. In view of the danger of leaving such a scar and since she had five living children, a sub-total hysterectomy was done. Both the patient and her new baby did well afterwards and were discharged from the hospital in good condition. She was seen in the office on May 4, June 1, and August 3, 1955, and was found to be doing very well.

Comment

The fetus in the case presented measured 8 inches or 20 centimeters, approximately eighteen weeks (menstrual age) gestation. The slight degree of maceration, however, might indicate that death had occurred less than three weeks before delivery.

Novak[10] states that the three pathological changes which characterize hydatidiform mole are: (1) trophoblastic proliferation, (2) hydropic degeneration of the villous stroma, and (3) scantiness of blood vessels. Some of the villi in the case reported showed these changes microscopically, as well as being grossly cystic and hydropic. Others were normal in appearance. The morphologic character of the specimen with the clinical picture of a fulminating pre-eclampsia at twenty-one weeks gestation makes the diagnosis of a hydatidiform mole. However, since there are some normal villi present and since there was a fetus present, it is a transitional hydatidiform mole rather than a typical or classical one.

Chesley, Cosgrove and Preece[4] state that 10 to 40 per cent of moles are complicated by a true toxemia of pregnancy and quote Hitschmann as saying that 30 per cent incidence of toxemia is about average. Compared with Eastman's[6] 6 to 7 per cent incidence of toxemia of pregnancy in general, it is obvious this complication is much more frequent with molar pregnancies. In fact, probably one should be suspicious of a hydatidiform mole when a fulminating pre-eclampsia develops in the first half of pregnancy. Chesley, Cosgrove and Preece,[4] in reviewing thirty-six cases of molar pregnancy accompanied by eclampsia, noted that there was a definite fetus found in seven cases. Melody[8] reported a case of hydatidiform mole with a four months' size fetus and complicated by a fulminating toxemia, very similar to this author's case. Melody's case was treated by interrupting the pregnancy by means of an anterior vaginal hysterotomy. The villi in his case also showed some to be normal while some were hydropic without vessels.

In the article by Hertig and Edmonds[7] it will be noted that approximately one-fourth of the transitional moles had well-formed fetuses present. Hertig stated he has seen only one case of hydatid degeneration of the placenta with a fetus of more than twenty-eight weeks. Cline[5] reported a case of a mole with a six to eight week fetus present, Mueller[9] had a patient who delivered at thirty-fours weeks, a 3 pound 14 ounce baby with a placenta in which there was 75 per cent involvement in hydatidiform degeneration. Mueller also gave references to five other similar cases. Beltz and Hutchins[1] reported a patient who at thirty-seven weeks gestation was delivered by cesarean section of a 6 pound ½ ounce living child with a placenta previa accompanied by a separate mass of molar tissue. It is probable this may have been a twin pregnancy. Also, Bowles[3] reported on a patient who developed a mild pre-eclampsia at seven and one-half months and was induced. She delivered a normal living baby of 4 pounds 6 ounces. Following this she delivered a 4 pound 6 ounce hydatidiform mole also. In this report Bowles quotes Meyer as finding in a collection of moles at the Carnegie Institute of Embryology, that 66.4 per cent of them contained a fetus in an excellent state of preservation. Their average age was 66.6 days.

Summary

A case of transitional hydatidiform mole with severe fulminating pre-eclampsia developing at twenty-one weeks' gestation, and accompanied by a well formed fetus of approximately eighteen weeks' size has been presented.

A short discussion has been given on the incidence of hydatid degeneration and the incidence and character of the transitional variety. Approximately 4 per cent of all pregnancies are involved in hydatidiform degeneration of some sort and of these 1.44 per cent are transitional moles. About 25 per cent of these transitional hydatidiform moles are present with a well formed fetus. Apparently in these cases the fetus lives as long as there is enough normally functioning placental tissue to sustain it. The in-

(Continued on Page 412)

Seminar

THE PYRROL PIGMENTS AND HEMOGLOBIN CATABOLISM

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(Continued from May Issue)

Serum Bilirubin and the van den Bergh Reaction

Since the epoch-making discovery of van den Bergh, Snapper and Mueller in the application of the Ehrlich-Pröscher diazo reaction to the quantitative determination of the serum bilirubin and, what has proven of greater interest, the different types of reactions which occur with different forms of jaundice,[39] there has been a great deal of study of the fundamental basis of these differences, despite which there is still disagreement and lack of exact knowledge.

It is well known that van den Bergh observed a prompt direct diazo reaction with bile in general, or blood serum from patients with jaundice in which it was reasonable to suppose that bile was being regurgitated into the blood. In this connection van den Bergh spoke of obstructive jaundice, but it is clear that this applies to intrahepatic cholangiolar disturbance as well as ordinary mechanical obstructive jaundice of extrahepatic type. In contrast, the serum from patients with pernicious anemia and hemolytic anemia, and normal horse serum, which contains a relatively large amount of bilirubin, all gave an indirect or delayed type of reaction. Van den Bergh could not decide whether these different reactions were due to some minor chemical variation in the molecule itself or to some substance in the bile causing the bilirubin to react in a prompt direct fashion, although he favored the former explanation. If the latter, it was necessary to assume that the effect was disspelled by heating, as boiling for five minutes caused the bile to give an indirect reaction. If the former, it was necessary to assume that chemical treatment leading to the crystallization of bilirubin from bile had converted it to the indirect reacting type as the same crystals were obtained, albeit by different methods, from indirect reacting serum on the one hand and from bile or prompt direct reacting serum on the other. In this connection it should perhaps be emphasized that a different method was necessary in the latter case because of the ease of oxidation of the prompt direct reacting bilirubin. The reason for this is not yet clear, but it constitutes one of the well-established differences between the two forms. It may well be related fundamentally to the greater water solubility of the prompt direct type and the correspondingly greater solubility in chloroform of the indirect type. In addition to these differences the two forms have been found to differ significantly in their absorption spectra.[41] There has been general confirmation that crystalline bilirubin isolated from cattle gallstones or bile, or as more recently obtained in this laboratory from human feces,[51] is identical with the crystalline bilirubin formed in old hemorrhages, as initially described by Virchow. Here the most decisive link was the crystallographic study of Fischer and Reindel,[13] as already mentioned. This clearly established the identity of the two forms. Since their source of extravasated blood was an echinococcus cyst of the liver the bare possibility had to be considered that the bilirubin was in some way derived from liver bile and not from the extravasated hemoglobin. Rich and Bumstead[42] later identified the crystalline bilirubin isolated from a hemorrhagic mesenteric cyst with that obtained from cattle gallstones.

Despite a voluminous literature and many concepts as to the nature of the van den Bergh reaction, little advance has been made until very recently. The studies of Cole and Lathe[43,44] in England have provided concrete information of great value. Before considering these studies in more detail, it is perhaps desirable to comment briefly

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The first E. T. Bell Lecture of the Minnesota Pathological Society.

From the Department of Medicine, University of Minnesota Medical School and Hospital, Minneapolis. The studies in this laboratory have been supported in part under contracts with the Surgeon General's Office, U.S.A., and the Atomic Energy Commission. Presented in part at the meeting of the Minnesota Pathological Society, October 18, 1955.

This lecture will appear in three installments, of which this is the second.

on the possibility that a different binding might explain the difference between the prompt direct and the indirect or delayed reaction. Various investigators had suggested that the indirect reaction was due to a combination of bilirubin with a serum protein, either albumin, globulin or globin. Of the three the latter was perhaps the most logical assumption, and that which was hitherto favored in this laboratory.[26a] The term "bilirubinglobin" was first suggested by Duesberg,[45] who believed that in the formation of bilirubin from hemoglobin and prior to the excretion of bilirubin by the liver cells, the bilirubin as first formed was still attached to globin. Fiessinger and co-workers[46] reported that a combination of bilirubin and globin, *in vitro,* gave an indirect reaction while bilirubin and albumin gave a direct reaction. Since it was well known from the work of Snapper and Bendien,[47] Pederson and Waldenström,[48] and Coolidge[49] that all the bilirubin in the serum, whether direct or indirect, traveled with the albumin fraction, either electrophoretically or in the ultracentrifuge, it would have been necessary to assume that if the indirect reacting form were actually a bilirubinglobin, the relatively small amount of globin involved would be masked by the larger amount of the serum albumin. The similar molecular weights and ultracentrifugal behavior made this theoretically feasible, also there was the possibility of a molecular complex between the small amount of globin and some fraction of the serum albumin. One of the main objections to this theory has already been mentioned—that free crystalline bilirubin gives an indirect reaction. This, however, did not exclude the possibility that the behavior of crystalline bilirubin with the diazo reagent is on a different basis than that of the indirect reacting bilirubin in serum.

The work of Cole and Lathe[43,44] fully confirms van den Bergh's observations and belief in the essential difference of the two types of reactions, but does not support the concept that the delayed or indirect type is bilirubinglobin. In brief, they were able to separate the direct and indirect reacting types by reverse phase chromatography, using silicone coated Kieselguhr. It was evident in their study that the direct type was polar in its behavior, that is relatively much more water soluble; the indirect type non-polar, or less water soluble. The indirect type obtained in this fashion was not associated with a protein. Its non-polar character is quite in accord with the earlier observations that it is much more readily extracted by chloroform;[40,50a] indeed, it is highly likely that all of the differences in behavior of the two types are based on relative water solubility. Cole and Lathe[43,44] consider that the two direct or polar types of pigment which they find in fresh bile and in blood serum from patients with regurgitation jaundice, differ chemically from the indirect type. They regard the latter simply as free bilirubin and believe that the direct type is converted to the indirect or to free bilirubin by various chemical manipulations, to account for the fact that one can isolate crystalline free bilirubin from bile, gallstones or human feces.[51] As van den Bergh and Snapper showed years ago, crystalline bilirubin can also be isolated from direct diazo reacting human serum,[39] but this requires a somewhat different and more complicated procedure than is true with indirect reacting serum. Cole and Lathe's concept that the direct reacting bilirubin differs chemically from the indirect is in essential agreement with van den Bergh who believed that a chemical change occurred on heating the bile or during the extraction process in which crystalline bilirubin is obtained from cattle gallstones, since heating is employed in that process. While it has not yet been proved that the indirect reacting bilirubin of heated bile is identical with that of hemolytic jaundice serum, or for that matter with the crystalline bilirubin as isolated from any one of a variety of sources already referred to, it is reasonable to assume at least tentatively that they are identical. On the basis of the conversion from direct to indirect by heating, van den Bergh, and others after him, considered that the direct type might represent a complex with one or more of the bile acids, but adequate evidence for this has not been obtained. Further study is needed with respect to possible complexing of bilirubin with some naturally occurring metabolite, to explain the prompt direct reaction, also the polar behavior and urinary excretion which is correlated with it.

It would lead too far afield to treat in any detail of the significance of the fractional serum bilirubin in clinical medicine, but a few words may be desirable. Although, due to lack of a sensitive photometer, van den Bergh made no attempt to determine the different types of bilirubin quantitatively, he clearly recognized that a prompt character was the most significant feature of the direct reaction and that the delayed direct increment was fundamentally related to the indirect type. Our

own experience over the years has been in full accord with this concept. In cases of pure retention jaundice, as for example, simple hemolytic jaundice or constitutional hepatic dysfunction from the delayed components. If one postpones measurement of the direct reacting bilirubin to thirty minutes, as in the Evelyn-Malloy method,[52] the distinction of pure retention jaundice, such as

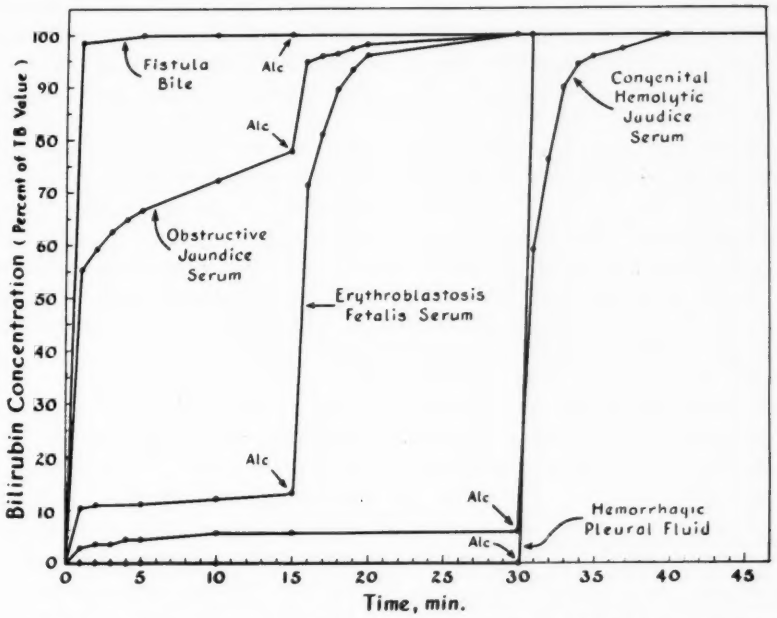

Fig. 5. Time curves of diazo reactions of various body fluids and jaundiced sera. (From Zieve, L., Hill, E., Hanson, M. L., Falcone, A. B., and Watson, C. J., *Journal of Laboratory and Clinical Medicine*, vol. 38, p. 446, 1951. Reproduced by permission of the publishers.)

(Gilbert's disease), the serum exhibits little or no prompt direct reaction. Often none whatever can be observed with the naked eye, at least within the first few minutes. On standing for a time, however, a definite slowly developing "delayed direct" reaction usually appears. If alcohol is added at any time during this period, all of the bilirubin reacts quickly in the characteristic indirect reaction. The lack of direct reaction is even more striking in hemorrhagic serous effusions in which local formation of bilirubin has occurred (Fig. 5). As van den Bergh emphasized, the prompt direct reaction of bile or serum of patients with regurgitation jaundice occurs within thirty seconds to one minute. It was on this basis that Ducci and I first advocated measurement of the direct reaction at one minute.[50a] Study of the time curves of the direct reaction of many different samples of blood serum from various diseases indicates that while the one minute measurement is arbitrary, it serves well in separating the prompt uncomplicated hemolytic jaundice or constitutional hepatic dysfunction of the Gilbert type, is not as clear. Thus in a case of chronic familial hemolytic jaundice the one-minute direct value was 0.3, the thirty-minute 1.8, the total (after alcohol) 4.8. There is every reason to believe that the thirty-minute value included a significant fraction of the non-polar or indirect type, which has reacted in a delayed direct fashion. It is quite possible that this is true also of a part of the one-minute fraction, but at least the proportion which reacted was so small that the contrast was striking and quite in accord with the lack of bilirubinuria and the concept of a pure retention jaundice.

For a time Ducci and I[50a] used a fifteen-minute measurement in addition to that at one minute, but this is believed unnecessary and experience has shown that one obtains the information of value from the one-minute determination and from the total following the use of alcohol, as in the regular Evelyn and Malloy modification[52] of van den

Bergh's method. It may be noted in passing that the essence of the Evelyn-Malloy modification was a primary dilution of the serum to a point where alcohol could be added without precipitation of the serum proteins. Van den Bergh had already recognized that some loss of bilirubin occurred on the precipitated protein, but his colorimetry was not sufficiently sensitive to permit a primary dilution of the serum. With the development of the Evelyn photoelectric colorimeter this became possible and constituted the valuable modification which Evelyn and Malloy proposed and which has come into general use.

In our hands and in many other clinics, measurement of the one-minute, or prompt direct reacting fraction and of the total bilirubin and their ratio, has enhanced the value of the serum bilirubin determination. It is fully evident that if the determination is restricted to the total bilirubin as in some laboratories, information of value will often be quite neglected. In brief, the particular information that one can expect to obtain may be listed as follows:

1. Significant increase of the prompt direct fraction within a normal total. The latter has a rather wide range from .5 to 1.5 per 100 cc. A study with Zieve in this laboratory[53] revealed an upper limit of normal for the one-minute fraction of 0.25 mg. per 100 cc., most often less than 0.2. While the determination of the prompt direct or one-minute fraction is relatively inaccurate, it is quite adequate to distinguish normal and abnormal values, as shown in the study with Zieve. In many cases of infectious hepatitis, either in the preicteric stage or in instances in which jaundice never develops, the one-minute fraction may be found between .3 and .8 with a total value less than 1.5.

2. The smaller the proportion of prompt direct reacting bilirubin, the greater the preponderance of retention as versus regurgitation jaundice. This is represented in its purest form by uncomplicated hemolytic jaundice, or non-hemolytic, constitutional hepatocellular dysfunction. In the former the excess of serum bilirubin is presumably due to overloading, in other words, overproduction beyond the ability of the liver to excrete bilirubin, and in the latter to a constitutional sluggishness or specific disturbance of the liver cell in the excretion of bilirubin. In such instances the proportion of prompt direct reacting bilirubin is below 20 per cent. Such a finding at once indicates the presence of a retention jaundice and is of immediate value in the primary classification of the problem. It should be mentioned that in recent years another type of constitutional hepatic dysfunction has been described and has come to be known as Dubin's disease.[45] The exact nature of this disturbance is still not clear. The liver cells are filled with a dark brown pigment, as yet unidentified. In this disease, however, regurgitation jaundice is characteristic and the percentage of prompt direct reacting bilirubin is much larger, usually from 50 to 70 per cent even at levels of total bilirubin in the range of 3 to 6 mg. per cent. The contrast between these two forms of constitutional jaundice in itself offers decisive evidence of the value of the fractional serum bilirubin determination.

3. In the presence of regurgitation jaundice with bilirubinuria, the finding of a relatively low percentage (20 to 40) of prompt direct reacting bilirubin, in our experience points strongly toward a diffuse hepatic disease with an associated hemolytic activity. This is seen in some cases of hepatic cirrhosis and at times in subacute or chronic hepatitis. It may be observed also in Hodgkin's disease or leukemia with hepatic involvement, splenomegaly and jaundice, also hepatic carcinomatosis.

The fractional serum bilirubin determination has gained a certain amount of undeserved ill repute because it commonly fails to differentiate between the regurgitation jaundice due to diffuse liver disease and that due to extrahepatic biliary obstruction, in other words between "medical" and "surgical" regurgitation jaundice. This is due to the fact that in many of these cases the overlapping of bilirubin ratios in the range from 40 to 70 per cent is too great to permit distinction. This is not surprising if one simply contemplates that in the hepatic types of regurgitation jaundice injury of the cholangiolar system within the liver is probably responsible for a return of bile to the blood and consequently for a relatively high proportion of prompt direct reacting bilirubin. This concept is at least strongly supported by the majority of available evidence. In our experience the cut-off point with respect to a reasonable accuracy in differential diagnosis, that is, in recognizing jaundice due to diffuse liver disease or retention jaundice on the one hand as contrasted with regurgitation jaundice due to obstruction, is about

35 per cent. Below this figure one can be fairly certain that one is not dealing with extrahepatic biliary obstruction. Values from 35 to 40 per cent are highly indicative, and from 40 to 45 per cent more suggestive of parenchymal or parenchymal-hemolytic jaundice, while above 45 per cent the serum bilirubin determination fails to be of distinguishing value. This is in general agreement with Ducci's observations, in a large material.[50b]

Excretion and Fate of Bilirubin

There is general agreement that under normal circumstances all of the bilirubin formed from hemoglobin catabolism is excreted in the bile and the majority of evidence indicates that little or none of it is reabsorbed from the intestinal tract. There is only fragmentary data as to the normal amount of bilirubin in the twenty-four-hour bile of the human being. Perhaps the best data on this point were obtained by Eppinger[22] in two patients with complete external biliary fistulas, both being in relatively good condition at the time of the observation. The spectrophotometric method which was employed was at least reasonably accurate. The values obtained ranged from 300 to 370 mg. per day. These are of considerable interest when contrasted with the lower values for fecal urobilinogen which will be discussed in a moment.

Many studies have shown that bilirubin is relatively unchanged until it reaches the cecum and ascending colon where the major reduction to the urobilinogen group takes place. This will also be considered in the following. First, however, it is desirable to comment briefly on the question of occurrence of bilirubin in the feces. It is well established that in the newborn period there is no reduction of bilirubin to the urobilinogen group, and according to With,[23] urobilinogen and urobilin are not found during the first three weeks, after which they steadily increase in amount so that after a year in the normal individual bilirubin is not demonstrable by ordinary methods. Fischer and Libowitzky[54] isolated crystalline bilirubin from the feces in a case of hemolytic jaundice. T. With[23] concluded that except for great overloading, as in hemolytic jaundice, bilirubin does not occur in the feces of normal adult humans, although it may appear in states of severe diarrhea. Studies in this laboratory with Sborov and Jay[55] clearly revealed that aureomycin and other broad spectrum antibiotics at least temporarily interfere with reduction of bilirubin in the colon with the result that it appears in considerable amount in the feces. By taking advantage of this observation and a new method of isolation, Lowry and the author were able to obtain relatively large amounts of crystalline bilirubin from the feces of human cases of hemolytic jaundice.[51] This has an important bearing on the urobilinogen problem and will be considered again in the following. Recent studies with Dr. Malcolm Campbell and Miss Hawkinson† have shown that bilirubin (and/or mesobilirubin) is regularly present in normal human feces in small amounts ranging from 5 to 20 mg. per day. The values in patients with hemolytic jaundice are considerably higher, as might be expected. From this it is evident that any study of total, hemoglobin derived pigment excretion must include bilirubin as well as its derivatives, which will now be discussed.

Urobilin and Urobilinogen Group

The term group is used advisedly because it has become increasingly clear that at least three compounds contribute in varying measure to the reactions which have been used in the past to characterize urobilin and urobilinogen. It will be evident in the following brief historical survey that the terms urobilin and urobilinogen are best suited to the designation of the *group*. From the time of the discovery of urobilin in 1868[56] until the crystallization of mesobilirubinogen by Hans Fischer in 1911,[57] information as to the nature of urobilin and urobilinogen was very vague indeed and in fact it was not until the isolation of crystalline stercobilin in 1931[67] and its subsequent comparison with the urobilin obtained from crystalline mesobilirubinogen, that the existence of at least two members of the urobilin group was clearly established. It will not be necessary here to review the literature of the development of our knowledge of this group in any detail. The older literature was admirably brought together by Meyer Betz in 1913.[58] In the author's Harvey lecture in 1948[59] an attempt was made to consider the more important work of the intervening years. Torben With's monograph on the bile pigments published in 1954[23] presents an extensive discussion of the literature. It may be helpful now to bring together in tabular form some of the more significant mileposts of progress in this long history.

†To be published.

Discovery of urobilin in urine and bile—Jaffé—1868.[56]

Identification of similar compound from feces, named stercobilin—Van Lair and Masius—1871.[60]

Amalgam reduction of bilirubin *in vitro* with resultant formation of urobilin type compound ("hydrobilirubin"); recognition that bacteria can similarly reduce bilirubin. Concept of enterogenous nature of urobilin—Maly—1871-2.[61a,b]

The enterogenous concept strongly supported. Transitory appearance of urobilin in feces and urine of case with complete biliary obstruction, upon feeding of urobilin free swine bile—F. v. Müller.[62a,b]

Clear recognition and naming of the chromogen urobilinogen—LeNobel—1887.[63]

The finding of a positive aldehyde reaction in the urines of many cases of liver disease—Paul Ehrlich—1901.[64]

Recognition that the aldehyde reacting substance of Ehrlich is urobilinogen—Neubauer—1903.[65]

Isolation of crystalline mesobilirubinogen after amalgam reduction of bilirubin—Hans Fischer—1911.[57]

Isolation of a crystalline urobilinogen from human urine; identification with crystalline mesobilirubinogen —Fischer and Meyer Betz—1911.[66]

Synthesis of mesobilirubinogen—Fischer and Adler.[66a]

Isolation of crystalline stercobilin from feces and urine —Watson—1931-33.[67,68,69,70]

Amalgam reduction of stercobilin yields a chromogen differing from mesobilirubinogen—Watson—1932.[68,69]

Isolation of crystalline urobilin IX,a* following oxidation of mesobilirubinogen *in vitro;* distinction of this urobilin from the stercobilin isolated from the feces and urine—Watson—1934[71]; Lemberg—1935.[72]

Synthesis of urobilin IX,a*—Sedel and Meier—1936.[71a]

Stercobilin strongly levorotatory, urobilin from mesobilirubinogen optically inactive—Fischer, Halbach and Stern—1935.[73]

Isolation of a dextrorotatory or d-urobilin from infected fistula bile—Schwartz and Watson—1942.[74]

Isolation of d-urobilin from feces of patients receiving broad spectrum antibiotics—Sborov, Jay and Watson—1949.[55]

Conversion of mesobilirubinogen to stercobilin—Watson, Sborov, and Schwartz—1942.[75]

Conversion of N_{15} labeled mesobilirubinogen to stercobilin—Lowry and Watson—1954.[76]

Isolation of crystalline d-urobilinogen and mesobilirubinogen from human feces—d-urobilinogen isomeric with dihydromesobilirubin—Lowry and Watson—1955.[77]

Conversion of d-urobilinogen to mesobilirubinogen by amalgam reduction—Lowry and Watson—1955.[77]

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*IX,a simply designates a configuration corresponding to protoporphyrin IX of hemoglobin, the porphyrin ring opening at the a or α methene bridge to form bile pigment. Many other urobilin isomers are theoretically possible and a few have been synthesized, but none other than IX,a have been found in nature. IX,a applies as well to the l- and d-urobilins and should not, therefore, be considered to distinguish the i-form.

It is simple and logical to designate the three members of the urobilin group on the basis of their remarkably differing optical activity: d-urobilin ($[\alpha]^{d}_{20^\circ}+5000$), i-urobilin (inactive), and l-urobilin or stercobilin ($[\alpha]^{d}_{20^\circ}-4000$). The latter is readily distinguished from the i- and d-forms by the Lemberg ferric chloride test,[78] which is a modification of Fischer and Niemann's method[79] of preparing mesobiliviolin from mesobilirubinogen. With the Lemberg test d-urobilin usually exhibits a green-blue or aqua color, i-urobilin blue or blue violet while l-urobilin is brownish yellow or at most slightly pink. The spectral characteristics of the final colored solutions are very helpful in distinguishing mixtures. Newer methods of crystallizing the urobilins, applicable to microgram quantities, have recently been described.[80,81] Also a new and relatively simple method permits preparation of i-urobilin from bilirubin, over mesobilirubinogen, but without concentration or crystallization of the latter compound.[80]

The exact role of d-urobilinogen in the normal pathway of reduction of bilirubin to stercobilinogen is not yet clear, but the above-mentioned study with Lowry[77] indicated the possibility that it may be a normal intermediary ordinarily reduced further by the normal intestinal flora. In occasional instances d-urobilin has been demonstrated in either feces or urine in which there was no history of the use of broad spectrum antibiotics or where these had not been used for a long period. In respect to the latter, however, an effect of long but variable duration must be recognized, and the possibility must also be considered that d-urobilinogen is formed only in the presence of an abnormal bacterial flora. Further work on this question is in progress.

It will be noted that the above survey takes cognizance of but one, the enterogenous concept of urobilinogen group formation. This has been widely accepted; indeed it appears incontrovertible on the basis of a great many studies, including those of Friedrich Müller, McMaster and associates and many others as set forth in the abovementioned reviews by Meyer Betz,[58] Watson[59] and With.[23] In these a discussion is also found of other concepts, notably the hepatogenous and the histogenous. In recent years, Baumgärtel of Munich has

advanced a dichotomous concept which has received considerable attention, especially in Europe, and therefore requires some discussion. This concept has not been included in the above survey, because as will be evident in the following, it is in serious conflict with many other well established facts and in the author's opinion it is not borne out by other significant observations, both prior and more recent.

According to Baumgärtel,[82,83,84] mesobilirubinogen is made only in the liver, or more properly by an enzyme in stagnant hepatic bile; stercobilinogen, only by bacterial activity in the feces. Baumgärtel has often asserted that mesobilirubinogen is not and cannot be converted to stercobilinogen, this despite the earlier studies in this laboratory as mentioned in the above survey as well as the more recent study with Lowry, in which it was clearly shown that mesobilirubinogen is converted by bacterial activity to stercobilin.[75,76] According to Baumgärtel and his followers, notably Stich[37,85] and Rudolph,[87] the optimum condition for mesobilirubinogen formation by the liver is a liver injury with associated stasis of bile. The characteristic absence of urobilinogen in urine and feces in the presence of complete biliary obstruction, or severe liver injury with complete suppression of bile flow, is explained by Baumgärtel as a "block" of the necessary enzyme. He speculates that when the serum bilirubin level is sufficiently elevated for the given individual this automatically turns off the hepatic dehydrogenase activity which is necessary to the formation of mesobilirubinogen.

However, this completely disregards the earlier failure of Elman and McMaster[88] to find any urobilinogen formation in their dogs with total bile fistula, in the absence of biliary obstruction, even in the presence of varying types and degrees of liver injury, nor does it take into account the striking exenteration experiments of Heilmeier and co-workers.[89] In these, the total removal of the intestine in dogs was followed by a complete disappearance of urobilin from the urine and there was no reappearance even when laked blood or hemin were injected parenterally, although in control animals such an injection was regularly followed by increase of urobilin in the feces and urine. Although Baumgärtel considered these experiments, it is difficult to understand on what basis he drew the conclusion that they prove his thesis, that of the hepatogenous origin of mesobilirubinogen.

Billi and Heilmeier[89] observed that urobilin and *indican* reappeared in the urine of these exenterated animals nine or ten days after operation, but only following obvious onset of putrefaction in the stomach and duodenal blind loop. Baumgärtel interprets the reappearance of urobilin in the urine at this time as due to an enzymatic formation of mesobilirubinogen in the stagnant bile, but this scarcely explains the simultaneous reappearance of indican and of both urobilin and indican in the vomitus, clearly related, as Billi and Heilmeier emphasized, to bacterial putrefaction. More recently, W. C. Meyer[86] studied a case of small intestinal fistula in which it was noteworthy that members of the urobilin and urobilinogen group were entirely lacking from feces, small intestinal contents of the fistula, and bile obtained by duodenal drainage. Baumgärtel has stated repeatedly that sterile "liver brei" is capable of converting bilirubin to mesobilirubinogen *in vitro*. We have not been able to confirm this using either sterile rat liver or surviving rat liver excreting a relatively normal bile for eight to twelve hours.‡ Nevertheless it is conceivable that under certain peculiar conditions some reduction might take place even in the absence of bacteria. This would scarcely prove that the liver was the only or even the more important site of mesobilirubinogen formation *in vivo*.

Recent studies in this laboratory with Graham, Ziegler and Lowry,[90] revealing that bacteria readily carry out the transformation of d-urobilinogen to mesobilirubinogen and thence to stercobilinogen, together with the transitory disappearance of all three following aureomycin or terramycin therapy and their reappearance with time, in the order given, would be most difficult to reconcile with any but an enterogenous concept. Baumgärtel[91] has objected to the aureomycin studies on the speculation that aureomycin damages the liver and interferes with the enzymatic formation of mesobilirubinogen. The later report of Gohr and co-workers[92] was of interest in this connection. They found that urobilinuria (not stercobilin) persisted in cases of liver disease receiving aureomycin. The significance of d-urobilin was not considered, nor have Baumgärtel

‡Unpublished observations with S. Schwartz, G. Price and M. Campbell.

or other proponents of his concept included it in their considerations.

In our experience aureomycin or terramycin regularly produces a transitory disappearance of the urobilinogen group from the urine; and when it reappears, it is represented by the d- form, which, as already mentioned, is readily formed by the action of at least certain fecal bacteria on bilirubin, and is readily converted by bacterial or other reduction to mesobilirubinogen. It would not be surprising, however, if i-urobilin were to be found at some stage in such cases; indeed it would be anticipated on the basis of our recent observation that after discontinuance of aureomycin the feces for a time contains d-urobilin, later i-urobilin, and still later l-urobilin, or stercobilin. This observed sequence in itself is of the greatest significance with respect to the enterogenous source of all three. Baumgärtel and Zahn[91] stated that when large amounts of dihydrostreptomycin were given orally in a patient with cirrhosis of the liver excreting considerable amounts of urobilinogen in the urine, the feces became negative for urobilin or urobilinogen, while the urine remained positive. This was interpreted as a cessation of the bacterial formation of stercobilinogen with continued hepatic formation of mesobilirubinogen. Lowry and I* were unable to confirm this, as in our experiment dihydrostreptomycin in the same amount used by Baumgärtel and Zahn failed to affect the fecal flora sufficiently to cause disappearance of the urobilin group from the feces. This failure, indeed, was to have been expected on the basis of many other studies of the *in vivo* effect of various antibiotics on the fecal flora.

Bingold and Stich[86] have recently reviewed studies of the past few years which have sought to determine whether Baumgärtel's concept is supported by clinical observations of the relative amounts of i- and l-urobilin (stercobilin) in various disease states. Basing their conclusion mainly on the earlier studies of Stich[85] in which the pentdyopent and mesobiliviolin reactions were used, and on the subsequent findings of several other investigators using the latter of these two tests, they state that under normal circumstances and in states of increased hemolytic activity, also in pernicious anemia, stercobilinuria is characteristic, while in liver disease i-urobilin usually predominates, although they observed a mixture with stercobilin in some instances. They agree with Baumgärtel that when there is complete disappearance of the urobilinogen group from the urine in cases of severe parenchymal jaundice as typified by infectious hepatitis, the controlling factor is the blood bilirubin level. As this rises, it is conceived that the cellular fermentative conversion of bilirubin to urobilinogen in the damaged liver cell is increasingly depressed to the point where it disappears entirely. Interestingly enough, they observed stercobilinuria during the receding phase of jaundice in these cases, and failed to explain why, with the obvious persistence of liver functional impairment at this stage, they did not find i-urobilin.

Stich[85] claims that urobilinogen is often demonstrable in the urine and feces in patients with complete mechanical biliary obstruction. In our previous studies of this aspect of the urobilinogen problem, complete biliary obstruction, as most often due to cancer of the pancreas or biliary tract, is characterized by the presence of less than 5 mg. of urobilinogen per day in the feces, and less than 0.3 mg. in the twenty-four-hour urine. Based on earlier work, it has generally been thought that these trace amounts were derived from bilirubin liberated from colonic epithelial cells swept off by the feces, and that this bilirubin was then reduced in the usual fashion to the urobilinogen group. Stich, however, claims that in this situation only mesobilirubinogen or i-urobilin is represented, and he believes that the traces in the urine were formed in the stagnant bile above the obstruction, those in the feces, by the action on bilirubin of cellular enzymes in the intestinal epithelium. He apparently did not consider the possibility that bacterial reduction of bilirubin might progress only as far as d- or i- urobilin and not to the l-form (stercobilin) under the influence of the abnormal environment of bile exclusion. Some indication of this was found in this laboratory a number of years ago[75] in the observation that stercobilin was not formed from mesobilirubinogen (i-urobilinogen) *in vitro* on the addition of acholic feces, but was observed with normal feces.

Within the past two years studies have been carried out in this laboratory with respect to the relative amounts of i-, d- and l- urobilins in the urine in various types of disease, notably hemolytic states and liver disease. These have failed to support the view of Baumgärtel, Stich and others that there is a relatively strict separation between

*Unpublished observations.

these two general groups insofar as the proportion or i- and d- urobilin on the one hand, and l-urobilin on the other, is concerned. In brief, the crystalline material and the mother liquor were examined from both urine and feces in these cases and no consistent pattern was observed. In the hepatic cases just as much stercobilin was found as in those with hemolytic states, and vice versa with respect to i- and d-urobilin. The results of this study will be reported in detail elsewhere. These observations, together with the clear evidence that mesobilirubinogen is converted to stercobilinogen by the fecal flora and that the fecal flora under various circumstances convert bilirubin either to d-, i- or l-urobilin, also that mesobilirubinogen (i-urobilinogen) regularly occurs in the feces, strongly indicate that if the liver participates in urobilinogen formation, it must be to a very minor and unimportant extent and that in general the intestinal origin of the urobilinogen group is by far the most significant.

Addendum

Very recently R. Schmid* has found that the prompt direct bilirubin of bile and of the serum and urine in cases of regurgitation jaundice, is a glucuronide. Billing and Lathe** have independently obtained evidence that this is the state of the bile bilirubin. These observations go far in clarifying the nature of the differing van den Bergh reactions.

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cidence of toxemia in molar pregnancies is considerably greater than that of all pregnancies in general.

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Continuation Studies

THE DIAGNOSTIC USEFULNESS OF PERITONEOSCOPY

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Peritoneoscopy is a simple and convenient method of examining the peritoneal cavity and has proved to be a very helpful procedure in properly selected diagnostic problems.

Peritoneoscopy was originally done with cystoscopes which Kelling used to first demonstrate the technique at Hamburg, Germany, in 1901.[1] Since then the procedure has gradually gained popularity, particularly since 1934 when Ruddock introduced an improved peritoneoscope which is most generally used in this country today.[2,3,4]

In addition to inspection, a number of other procedures may be performed which greatly increase the usefulness of peritoneoscopy. Among these are biopsy, either by means of a special forceps through the peritoneoscope or with a liver biopsy needle separately introduced through the abdominal wall and guided to the desired area with peritoneoscopic vision.[5,6] Techniques for peritoneoscopic cholangiography[7,8] and measuring biliary pressures[9] have been described. Splenoportography has also been done under direct peritoneoscopic control.[10]

Indications

The chief indications for peritoneoscopy are unexplained ascites, liver disease and epigastric masses. Suspected carcinoma of the gallbladder or tuberculous peritonitis are less common but equally good indications. Since intravenous cholangiography still requires reasonably good liver function, it will fail with jaundiced patients in whom cholangiolitic hepatitis or biliary cirrhosis cannot be distinguished from surgically correctable biliary obstruction, and in such instances peritoneoscopic cholangiography has proved very useful. Lesions of the female pelvic organs are examined better by culdoscopy.[11]

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The Procedure

The actual technique of peritoneoscopy is simple, being only slightly more involved than the ordinary paracentesis. Preoperative measures should include bleeding, clotting and prothrombin times and 1 liter of blood typed and cross matched. A simple brief explanation of the purpose and nature of the examination is usually sufficient to obtain the consent and confidence of the patient. Preoperative medication depends upon the patient's condition; most will tolerate 75 to 100 mg. of demerol, but this is not used in patients having severely impaired liver function. Since many candidates have liver disease, barbiturates are seldom used.

The patient's abdomen is prepared with ether, tincture of zephiran and sterile drapes. The point of entry is selected usually lateral to, or just below, the umbilicus and infiltrated with 1 per cent xylocain down to the peritoneum. A small incision, about 0.5 inch, is made through which a trocar is introduced and a tense pneumoperitoneum established with carbon dioxide. The trocar is then replaced by a larger cannula which carries the peritoneoscope.

Structures seen include the anterior abdominal wall, lower portions of the liver, fundus of the gallbladder, lower pole of the spleen (if enlarged), anterior wall and lesser curvature of the stomach, omentum, loops of bowel, parts of the female genitalia and dome of the urinary bladder. Examination of the upper abdomen is facilitated by the Fowler position and that of the pelvic cavity by the Trendelenburg position.

If a biopsy is desired a special forceps with its own lens and lighting system is put through the cannula in place of the examining scope. These forceps will remove a piece of tissue slightly less than a cubic centimeter in size from the surface of the liver. In some instances, when a deeper specimen of liver is desired, a liver biopsy needle is introduced through the abdominal wall and its

tip, which is easily seen as it pierces the parietal peritoneum, is directed to the area desired for biopsy. A biopsy improves the accuracy of the diagnosis and is attempted whenever possible.

If the gall bladder can be seen, cholangiograms are easily obtained by introducing a 6-inch No. 20 needle through the abdominal wall overlying the gall bladder and then, under peritoneoscopic vision, piercing the bladder wall. Bile is aspirated and 5 to 25 cc. of 35 per cent diodrast injected. The needle is withdrawn and roentgenograms obtained at intervals to follow the course of the contrast material. Dehydrocholic acid, nitroglycerin or both may be given to facilitate passage of the diodrast into the duodenum.

When the procedure is completed, the CO_2 is expelled and the incision closed with a skin clip. The time required for inspection and biopsy is usually ten to fifteen minutes, an additional five to ten minutes is required for injecting the gall bladder. When the patient is returned to the ward he may be up and about and eat as desired. If a biopsy has been done he is required to stay in bed at least twenty-four hours during which hourly blood pressures and pulse are recorded. Postoperative analgesics are not needed but the patient is told about moderate shoulder strap pain which may result from air under the diaphragm and is no cause for alarm.

Contraindications

Abdominal distention from intestinal obstruction is an absolute contraindication to peritoneoscopy. An adequate pneumoperitoneum cannot be established and the danger of bowel perforation is prohibitive. The acute abdomen is also a contraindication. The rigid abdominal wall frequently prevents a sufficient pneumoperitoneum and the danger of spreading possible infection is too great. The patient's general condition is sometimes a contraindication such as the severely debilitated elderly subject with diminished vital capacity or advanced terminal disease.

Liver biopsies cannot be obtained if the patient has a bleeding tendency including elevated prothrombin times not responding to Vitamin K. Greatly increased biliary pressure also precludes biopsy since the wound may continue to ooze bile for days causing bile peritonitis, fistulae or bleeding. Lesions in the retroperitoneal structures such as pancreas, aorta, kidneys or retroperitoneal nodes cannot be seen and peritoneoscopy in these conditions is of no value unless liver metastases are being sought.

Complications

Abdominal adhesions are the most frequent complication causing failure of peritoneoscopy because they prevent adequate visualization. Attempts to divide or break up such adhesions with the scope invite the danger of intra-abdominal hemorrhage. Loops of bowel may be bound to the abdominal wall at the site of laparotomy scars and consequently the peritoneoscope should never be inserted through or close to previous incisions. Other occasional minor complications include subcutaneous emphysema localized about the incision or a small ecchymosis in this area. Such conditions are painless and disappear quickly. Bleeding from a liver biopsy is, of course, another complication which must be carefully watched for by close observation of the patient and the frequent recording of the pulse and blood pressure for a period of at least twenty-four hours.

Discussion

Peritoneoscopy is not intended as a substitute for an exploratory laparotomy. Each has its specific indications which are in most instances mutually exclusive. Since peritoneoscopy is a simple, convenient and safe procedure to which patients readily consent it is to be recommended when the proper indications, as discussed in the foregoing sections, are present.

Our experience has shown that the method has been diagnostic in over two-thirds of the patients for whom it was requested because of a diagnostic problem and contributed helpful information in an additional one-fourth.[12] Others have reported similar results.[13] Peritoneoscopy has been of particular value for patients in whom a prolonged prothrombin time precludes blind needle biopsy of the liver. In such patients, direct inspection of the liver will usually confirm or exclude the suspected diagnosis. As new advances and unsuspected innovations are made in medicine peritoneoscopy may well continue to acquire new uses, as it has in the past with cholangiography, biliary manometric studies and splenoportography.

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Current Concepts

LYMPHEDEMA

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Lymphedema is a relatively uncommon disease. Consequently, the practitioner of medicine does not have occasion to think of it often. In addition to this, the fact that lymphedema is a chronic and disfiguring disease which is refractory to treatment does not enhance its position in the mind of the physician. A third factor which tends to make us overlook this phase of peripheral vascular disease is the insidious onset of certain inflammatory forms, their presence being masked by the acuity or severity of the primary illness until the fully developed syndrome of lymphedema is apparent, well established, and oftentimes irreversible.

No attempt will be made to classify or to discuss all of the manifestations of lymphedema. We shall concern ourselves only with lymphedema praecox and with certain of the inflammatory forms of lymphedema in this short discussion.

Lymphedema praecox is the most frequently seen form of non-inflammatory lymphedema. Although it is seen in both sexes, it is much more common in the female. It usually has its onset sometime between late childhood and early adulthood and rarely begins before nine or after twenty-five years of age. This distinguishes it from the congenital forms of lymphedema (i.e. Milroy's disease and simple congenital lymphedema). It is characterized by the gradual development of edema of the affected member(usually unilateral) which may either be relentlessly progressive until the extremity is greatly deformed or which may stop its progression at any point. There is no pain except for the discomfort incident to the weight of the enlarged extremity. The past medical history is non-contributory and the family history is non-contributory. Although these patients may have infections of the feet secondary to the chronic edema, infection plays no role in the development of lymphedema praecox.

Physical examination reveals varying degrees of edema depending on the stage of the illness. It begins usually in the most distal part and extends proximally. In the early stages, the edema is usually soft and pits easily, but as the disease progresses and fibrosis occurs, the tissues become very firm. It should be emphasized, however, that lymphedema will pit even in the late stages if enough extracellular fluid is present. The appearance and texture of the skin are quite distinctive. It becomes coarse and indurated and has quite a different appearance from the skin of patients with long-standing venous stasis. Another important physical finding is that the venous pattern is normal in lymphedema praecox. Squaring of the toes has been described as a useful physical finding. In our experience, this has not occurred constantly enough in early cases to be helpful.

Other causes of venous and lymphatic obstruction, notably pelvic malignancy, must be carefully excluded by the physical examination.

The treatment of lymphedema praecox is discouraging. However, the earlier the diagnosis is established, the more satisfactory the results. Medical therapy has a twofold objective. First, the existing edema should be controlled and further edema formation prevented as completely as possible. This is accomplished by putting the patient to bed with the legs markedly elevated for three to seven days (until the leg is either back to normal size or until it stops receding in size) and then providing the affected extremity with an adequate elastic support. Weight reduction is also important in controlling the edema if the patient is obese. The second objective in medical therapy is control of fungus infection and acute cellulitis should they occur.

It should be emphasized at this point that neither of these objectives will be attained unless

the attending physician enlists the wholehearted co-operation of the patient. In lymphedema, as in most chronic diseases, it is the patient who treats himself from day to day. He must make the decision every morning as to whether he will apply his heavy, hot rubber bandage, wash his feet and dry between the toes, and stay on his diet that day. He probably will not do these things faithfully unless the reasons for doing them are given to him clearly and simply and the alternatives are also made known to him.

Other than unwillingness on the part of the patient to co-operate, the most common cause of failure of medical treatment is failure to use adequate elastic support. This is defined as support that is heavy enough to control the edema completely. Usually a solid rubber bandage is ultimately required. This is admittedly distasteful to the patient, particularly the female patient. However, it is felt that the consequences of the illness are even more distasteful from the cosmetic viewpoint.

The surgical treatment of lymphedema consists of a plastic procedure designed to reduce the size of the limb and make it more useful. The procedure is difficult and must be done in two or three stages. A prolonged morbidity is common. Extensive scarring is the rule, and it must be understood by the patient that continued use of elastic support will be necessary postoperatively. It is a useful procedure when the need for it arises, but it is never done early in the illness or as a procedure to prevent progression.

Inflammatory forms of lymphedema are probably of more common occurrence than the noninflammatory forms. The two most commonly seen forms in this area are those occurring in conjunction with the trichophytosis and recurrent superficial cellulitis in patients with long-standing chronic venous insufficiency and those occurring from the same causative factors in the absence of chronic venous insufficiency. As was mentioned above, the lymphedema in this group is often overlooked in its incipiency because the chronically swollen leg, the recurrent acute cellulitis, et cetera, tend to mask it. It develops very slowly and does not become obviously apparent until the complete syndrome develops. This type of lymphedema can be arrested at any stage if the causative factors can be controlled. The treatment here is foot hygiene, prevention of trichophytosis, prompt and intensive treatment of cellulitis when it occurs, and control of edema when chronic venous insufficiency exists as a complicating factor. Occasionally, it may be necessary to resort to intermittent prophylactic chemotherapy or antibiotic therapy if the recurrences of cellulitis are frequent enough and are not controlled by the more conservative measures.

In the inflammatory forms of lymphedema, as in the noninflammatory forms, the patient must be carefully instructed as to the nature of his disease if the physician is to gain his wholehearted cooperation.

SHARPER X-RAY PINPOINTING OF HEART ABNORMALITIES

A complex x-ray examination, selective angiocardiography, is helping to pinpoint heart abnormalities in infants and children amenable to corrective surgery, three Toronto, Canada, physicians have reported.

The procedure opacifies limited and predetermined portions of the heart and great vessels where these specific areas might not achieve absolute clarity by the venous angiocardiographic method, the three physicians added.

They are Dr. Richard D. Rowe, Dr. Peter Vlad and Dr. John D. Keith, all associated with the University of Toronto School of Medicine. Their report is published in a recent issue (March, 1956) of *Radiology*.

"Selective angiocardiography . . . is the method of opacifying limited and predetermined portions of the heart and great vessels. This procedure has been used by groups in Europe but at present its value has not been recognized widely on the North American continent," the Canadian specialists stated.

They also described the complex steps they follow in the procedure after first administering a slight anesthetic to the child, which induces a light sleep during the examination.

"Twenty-nine infants and twenty-one older children (all but six under five years of age) have been studied by selective angiocardiography in our report. No untoward reactions have been encountered in this group," they reported.

Other conclusions advanced in their article:

The venous route is particularly helpful in small cyanotic infants (blue babies) where the range of diagnostic possibilities is wider than at any other time.

Intelligent use of the selective technique implies knowledge beforehand of the particular malformation. Its value lies in making refinement of diagnosis possible rather than arriving at the diagnosis itself.

Clinical Notes

XYLOCAINE JELLY FOR TOPICAL URETHRAL ANESTHESIA

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ATTAINING satisfactory local anesthesia of the male urethra, an important aspect of urologic practice, is considerably facilitated by proper lubrication during instrumentation. Xylocaine jelly (Astra) is an aqueous solution of 2 per cent lidocaine hydrochloride and 3.5 per cent sodium carboxymethylcellulose, which utilizes this principle by combining a potent topical anesthestic agent in a lubricating base.

Xylocaine was synthesized in 1943 by Lofgren and Lundquist. Its safety and effectiveness as a topical urethral anesthetic have been attested by Haines and Grabstald (1949), Nesbit and Baum (1952) and Persky and Davis (1953). These authors cite several patients voluntarily stating their preference of Xylocaine to agents previously employed. A patient of Persky and Davis with transient postcystoscopic syncope represents the only untoward reaction to topical urethral Xylocaine in slightly more than 1150 reported instances of its use.

This report concerns 122 male patients upon whom 150 office procedures were performed (Table I). No premedications were given. Fifteen cubic centimeters of Xylocaine jelly were injected into the anterior urethra, massaged into the posterior urethra and retained for about five minutes by a penis clamp applied at the coronal sulcus. Eighty-two patients underwent cystourethroscopy. Conditions found in the fifty-two diagnostic procedures included benign prostatic hypertrophy, urethral strictures, posterior urethritis, vesical calculi, vesical tumors, chronic interstitial cystitis and infection following transurethral prostatic resection. Twenty-seven patients had bladder tumor rechecks, and one patient had a small vesical recurrence coagulated without incident. Sixty-eight dilatations were performed for urethral strictures, vesical neck contractures and posterior urethral infection. No toxic or untoward reactions were encountered.

TABLE I

Number of Patients		112
Cystourethroscopic Examinations		82
1. Diagnostic	52	
2. Bladder Tumor Rechecks	26	
3. Bladder Tumor Coagulation	1	
4. Urethroscopy and Urethral Dilatation for Stricture	3	
Dilatation Procedures		68
1. Urethral strictures	49	
2. Vesical neck contractures	17	
3. Posterior urethritis and prostatitis	2	
Total Number of Procedures		150

Comment

The cost of 15 cc. of Xylocaine jelly is approximately four times that of a comparable amount of cocaine. Does Xylocaine jelly possess advantages that justify this increased cost? The answer is in the affirmative for several reasons. More profound anesthesia is noted than with cocaine, allowing better relaxation and improved cystoscopic visualization of the posterior urethra and vesical neck. Postcystoscopic discomfort is virtually absent. Injury to mucous membranes has not been observed. Two patients with previous minor toxic reactions to cocaine experienced no difficulty with Xylocaine. Several patients expressed preference of Xylocaine to cocaine. We believe these observations adequately demonstrate its usefulness as a topical urethral anesthetic.

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Editorials

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A CONTRIBUTION OF CLINICAL PSYCHOLOGY TO PSYCHOSOMATIC MEDICINE

One of the major roles of the clinical psychologist in the field of psychosomatic medicine is that of the research investigator, attempting to verify whether or not significant relationships exist between certain disease states or medical syndromes and certain personality characteristics or behavior patterns. Because the clinical psychologist is trained in experimental design and research methodology, as well as in clinical methods, he has attempted to place many of the armchair hypotheses concerning psychosomatic disorders under scientific and objective scrutiny. He has also been critical of much of the alleged evidence that certain physiological disorders have definite and specific psychological correlates because in most studies: (1) the general character of the populations possibly lacks representatives in the sense that few cases are used, and the socio-economic status, age, sex, race, duration of illness, the amount of restricted activity due to illness, and other variables usually represent a small selected segment of the general population; (2) there is grossness of the observational methods used along with lack of attempts at quantification and demonstrative reliability of the data; (3) there is generally an absence of control groups; (4) there is sometimes a lack of experimental or clinical reproductivity because of the influence of specific theoretical points of view.

Because many investigators use a rather loose and unquantified language, it makes it difficult to check various assertions with known psychological techniques. For example, it is stated that persons having asthma are dependent and unconsciously fearful of separation from mother; hypertensive persons are submissive with chronic and inhibited hostility; peptic ulcer patients are overly ambitious and independent as a means of compensating for deep dependency needs. There is a possibility that perhaps present-day techniques used to evaluate such statements are not sensitive enough to measure such rather complex unconscious motivation. However, psychologists have attempted to restate many of these hypotheses in testable form for objective and scientific study with the techniques they currently have at their disposal. As a result, many of the previously held specificity theories of psychosomatic involvement are beginning to fall by the wayside. In a subsequent editorial, examples of psychosomatic research by psychologists as individuals and as members of a medical research team will be discussed.

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ELECTROCARDIOGRAPHY: USE AND MISUSE

Electrocardiography has developed so rapidly that periodic evaluations of its usefulness are necessary. Overemphasis may stress its value beyond actual possibilities while underemphasis, misuse by poorly trained workers, or disappointment in its findings occasionally cause unwarranted criticism.

Technique.—Following an initial period of study, electrocardiographic technique was standardized, largely through the work of the school of the late F. N. Wilson. This technique consists of the use of photographic or direct writing apparatus and of twelve leads: three standard limb leads, three augmented V leads of the limbs (Goldberger's modifications), and six precordial V leads. Other leads or additional tracings should be taken by the physician himself in certain cases. They are: the high V leads in cases with possible anterolateral infarct; VE (xiphoid) and the posterior leads V_7, V_8 in cases with possible posterior infarct; V_4R and V_3R in order to detect right ventricular hypertrophy or focal block; V_3R and long strips of V_1 and aVF in atrial disorders (especially doubtful cases of atrial flutter); and long strips of lead 2 in cases with arrhythmias. Moreover, functional tests (including the two-step exercise test and the amyl nitrite test) may be useful in cases with precordial pain and normal resting ECG. Standard sizes of chest electrodes

(2.5 cm. for adults and 1.5 cm. for children) should be used.

Artifacts.—Numerous artifacts are possible and are usually easily detected by inspecting the tracing. Unfortunately, some of them may escape observation when the tracing is already mounted, especially if only a short section is visible. Among them, improper shape of the waves due to overshooting, overdamping, or improper calibration, are the most common.

Direct writing apparatus have justly become popular. However, improper functioning of the moving parts may add to the possible causes of error. Only frequent technical inspections and delicate handling guarantee an accurate reproduction of the patterns.

Mounting of a tracing is an easy chore which bores the most stolid technicians. Few of them appreciate that, in a few proven cases, normal persons have been kept in bed for weeks and submitted to unnecessary and enervating worry because a technician mounted a tracing upside down or substituted one lead (usually aVR) for another.

Application of the chest electrodes requires great care. Smudging of a large surface with electrode jelly may greatly change the pattern of the tracing. Use of normal instead of small chest electrodes in infants or children may obtain the same result.

It is well known that muscular currents may mar an otherwise perfect tracing and may even simulate the existence of auricular fibrillation. Few doctors know how to remedy this: the patients should lie down comfortably, not sit; their limbs should be placed at a proper angle; their mind should be put at ease; in cases of somatic tremor, round, flat electrodes applied over bony surfaces of the shoulders and pelvis may substitute with advantage the conventional electrodes.

Interpretation.—Certain data are of great value in the interpretation of a tracing. They are: age and sex; blood pressure, possible existence of valvular defects; drugs received by the patient; reason for requesting a tracing; and information supplied by previous tracings (or a sample of them). Comparison between subsequent tracings may be extremely important, both in cases of coronary heart disease and rheumatic carditis. Two subsequent tracings which are still within "normal limits" may reveal with their changes that a process in evolution is modifying the conditions of the myocardium.

The measurements of the P-R and of Q-T intervals are extremely important, particularly in cases of myocarditis.

In several diseases, information supplied by the electrocardiogram is inadequate and may even be misleading. Knowledge of this may be useful and indicate the need for a different method of study.

It is impossible to draw a sharp line between normal and abnormal tracings. In reaching a decision, the observer must consider the possible occurrence of a certain variation in the electrocardiogram of a normal person. Statistical tables are available and may be useful. However, personal experience and additional clinical and laboratory data help in determining whether an abnormality should be considered pathologic.

The most important diagnostic data are supplied by the ECG in the evaluation of atrial and ventricular rates, in the study of the electrical position of the heart, and in the evaluation of the conditions of the ventricular myocardium, especially for the study of changes of the coronary blood supply. Certain valuable data are also obtained in pericarditis.

It should be emphasized that the electrocardiographic patterns obtained in hypertensive heart disease, cor pulmonale, rheumatic valvular defects, and congenital heart diseases give only *indirect* evidence of the cardiac lesions and that evaluation of this evidence should be done carefully and with restraint.

In general, one should not try to draw unwarranted conclusions and to obtain from the reading of the ECG data which it cannot supply.

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THE CASHIER'S CHECK AND BANK DRAFT

Occasionally, in our personal or business dealings, an obligation is incurred which cannot be paid by remitting a personal check, but must be settled in cash or an acceptable substitute for cash. A familiar example of this type of situation is the closing of a real estate transaction. In circumstances where cash payment is demanded, the cashier's check and bank draft, although not legal

tender, are usually a readily acceptable substitute for cash.

A cashier's check is drawn on the bank from which it is purchased and for all practical purposes is that bank's I.O.U. It will be made payable to the order of any person or persons whom the purchaser may designate. Traditionally, this check has been signed by the person who holds the office of cashier of the issuing bank; hence, the name cashier's check.

Banks also sell bank drafts which are checks drawn against an account the selling bank carries with one of its banking correspondents. If the payee can be satisfactorily identified, a bank draft can be immediately collected in a distant city at the bank on which it is drawn. Since it is not the practice of banks to issue instruments which will not be paid when presented, the cashier's check and the bank draft have come to be widely accepted in lieu of cash payments. This wide acceptance of cashier's checks and bank drafts makes it possible for customers to make large or small cash payments in their own community or at distant points without the inconvenience or risk of manually transporting or shipping currency and coins.

CLARENCE G. FRAME, *Cashier*
The First National Bank
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THE NATURE OF ALCOHOLISM

"Whenever a disease is so widespread in the population, so serious in its effects, so costly in its treatment that the individual unaided cannot deal with it himself, it becomes a public health problem."—THOMAS PARRAN, former Surgeon General, U. S. Public Health Service.

Cases of alcoholism are seven times more numerous than those of tuberculosis. A conservative estimate places the number of alcoholics in the United States at almost 4,000,000. Nearly 1,000,000 of these people have suffered physical or mental deterioration caused by their excessive drinking.

Alcoholism ranks among the major public health problems in the country. Some authorities say it is the fourth largest.

Alcoholism is a symptom of emotional illness. Alcoholism is a substitute safety valve for emotional pressures, and it is used only because the "good" safety valves are not working as they should. *This gives us the clue to successful treatment or rehabilitation of the alcoholic person,* whether that treatment involves individual psychotherapy, group psychotherapy, A.A., pastoral counseling, or casework services. The good valves must be opened, and the substitute valves must be closed.

Alcoholism is no respector of persons, no caterer to class, color or creed. It can slip up on the doctor, the preacher, the painter or the housewife.

One thing all people suffering from alcoholism have in common is an uncontrollable need for the effects of alcohol. To that degree, at least, they are equally sick people. Both need treatment to halt the progressive development of their disorder. The alcoholic cannot do this by himself. *He needs help!* He desperately needs medical care, first to keep him alive, so that, secondly, the resources of medicine, the church, psychiatry and psychology supplemented by social welfare resources and attention can be brought into play to rehabilitate him as a productive and self-sufficient citizen.

PAUL S. RAHNEFF
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Fourth in a series of editorials on alcoholism.

THE HISTORY OF GLASS CONTAINERS

In the late pre-Christian era Egypt continued to produce glassware in increasing quantities, and her output was taken by Phoenician traders to ports throughout the Mediterranean. Essential oils, wine, honey and so on, once packed in pottery, were put into glass which was found infinitely more efficient. Chemically inert, then as now, the new glass containers added nothing to, and took nothing from, whatever was put into them. These first glass-packed products not only withstood long sea voyages, but long periods of storage as well. And, as they could be used again and again, the original cost was soon justified. As other countries became familiar with the protective advantages of glass, they too, tried their hand at producing it with the result that glass-making soon spread throughout the Mediterranean.

Rome, among others, cast covetous eyes on Egypt's thriving glass industry. In 30 B.C., when Augustus added Egypt to his empire, he demanded glass in tribute. Supplies failed to satisfy him,

Second in a series of editorials on glass and glass containers.

however, and he ordered Egyptian craftsmen to come to Rome to train others in their art. Because of Rome's patronage, the first four centuries of the Christian era are known as the First Golden Age of glass.

Following the fall of the Roman Empire, and the ensuing Dark Ages, Venice became the artistic and cultural capital of the world. Glassmaking was especially encouraged, and the Second Golden Age of glass began in Venice about the 13th Century and lasted for about four hundred years.

Like Rome in the First Golden Age, Venice had to import glassmakers at the outset. Artisans came from her colony in Syria, and were given royal treatment. But rich and respected as they were, these skilled glassmakers were also closely guarded and "protected," lest they—and their skills and trade secrets with them—be coaxed into other countries anxious to compete with Venice's glass industry.

Venice's star began to dim, however, and by the beginning of the 17th Century countries all over Europe were producing glassware, which equaled that of Venice in her greatest days. Bohemia became famous for her magnificent ruby glass. Glass factories were founded in Antwerp in 1550, in Brussels in 1662. Others sprang up in Germany and Holland, in Spain and France.

Glass factories were also blazing in England throughout the 16th Century. They could not, however, satisfy ever-increasing demands, and England was forced to import much of her glass. Further, coal had not yet come into general use, and the hungry fires of England's glass plants were depleting English forests. Officials of the London Company of Virginia accordingly turned their eyes toward America with the hope of establishing—among many other things—new and profitable glass factories on the wooded shores of the new continent.

Sent out by the London Company, a small and courageous band of colonists under the direction of Captain John Smith landed at Jamestown, Virginia, in 1607 to establish the first permanent English settlement in North America. A second group—which included eight glassmakers—arrived at Jamestown the following year, and, by the autumn of 1608, a glass factory was in operation in the nearby forest about a mile from the settlement.

JOHN M. FOSTER

TELEVISION: EVERYTHING FOR EVERYBODY?

"I'll buy a television set," the man said, "as soon as they start putting something really worthwhile on TV." You've met this man, possibly, although his ranks are thinning every day. The obvious question, of course, is how can he know anything about what "they" are putting on TV, if he starts out admitting he never sees it?

But there's another question, of much more significance to the nine out of ten people who *do* watch television. Does TV offer something of value in return for the time we give it?

The people most familiar with today's television are convinced it does. They realize television cannot suit every individual taste every minute of seventeen or eighteen hours each day. Neither television nor anything else can be everything to everybody.

Television's service to general knowledge comes directly in such programs as news, "See It Now," "Science in Action," "Omnibus," and a host of others—to name a few from the program schedule of WCCO-TV. Indirectly, television imparts valuable knowledge through such programs as "The $64,000 Question," "Hobby Showcase," "Highway Patrol." Even such sheer entertainment as "I Love Lucy," offers information through guests and comment on current events, social customs, and general contemporary life.

The average American home has been made into a center of information and entertainment not even the most wealthy could have enjoyed a decade ago. You and I—and our children—are familiar with the faces and voices of most of the great men of our age. We know the major cities and the great natural spectacles of the world. We've occupied front row seats at the top sports events; have watched great dramatic performances, operas, musical comedies. We've seen these things on our television sets—the next best thing to being at the scene in person. And past advances are only a prelude to future TV accomplishments.

In short, there are literally hundreds of TV features which may be watched with enjoyment and enlightenment by a literate adult, who may win mental stimulation and frequently a definite extension of his mental horizons. The best of them are as rewarding as anything you can introduce easily into your living room.

What's the answer to the fellow who says he won't watch television until "they start putting something really worth-while on TV?" Perhaps it's this: It's time to stop knocking TV's intelligence, and to recognize the surprising number of absorbing programs which truly add to general knowledge.

GENE GODT
WCCO-TV

MODERN PENOLOGY

Moderns recognize that man and his world are dynamic, not static. People and things change and no one has all the answers, but we should keep seeking the answers.

Penology (the study of how we handle criminals) has been changing since it got started. First, it meant "punishment." Later, out of necessity, it came to mean "correction." Penologists learned that mere punishment did not control criminal behavior. As moderns, they have sought for centuries better ways to combat crime.

This search has been inspired by all major religions and immeasurably aided by science. Medicine, through psychiatry, has helped to explain why we behave like human beings. Much has been learned from sociology, anthropology, jurisprudence and psychology.

Though society must properly hold the criminal responsible for his acts, it also distinguishes among criminals. As a result, we have witnessed the growth of hospitals for criminally insane, the juvenile court, youth commission and probation.

The penitentiary, as we know it, was established as a substitute for corporal and capital punishment. Like the other agencies mentioned, its job is to protect society. This it can do efficiently (albeit expensively) by keeping prisoners securely locked up. However, one cold fact makes this simple proposition meaningless because some *97 per cent of all prisoners eventually will be released.*

To truly protect society, prisons must do more than keep people locked up. They should try to prepare people for release to law-abiding lives. Given our present state of knowledge, this is not an easy task. But, do we really have any better alternative? Constructive work, sound discipline, case work, medical services, education, religion, and recreation all have a job to do in prison, with and for prisoners. When prisoners are released, it should be through the means of parole. This provides helpful, watchful supervision in the community toward the goal of a self-supporting, productive parolee.

Some 2,000,000 felonies are committed in the United States each year. We have the reputation of being the wealthiest and least law abiding of nations. Estimates vary, but all agree the annual cost of crime is enormous. Penologists and criminologists can apprehend, arrest, convict, and imprison offenders. The ratio of people imprisoned to felonies reported is surprisingly low. Some 80,000 people are sent to prison each year, or about one new convict for each twenty-five felonies. A large number of crimes go unsolved. Some criminals we fail to convict, others get jail sentences or probation. The best weapon against crime is prevention. This is the big job, the one that all citizens have to face.

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REHABILITATION IN PULMONARY TUBERCULOSIS

The word rehabilitation is now in such common use that it often threatens to become quite meaningless. It is used for various remedial exercises, for retraining of the fit, and vocational training to new work of those unfit for their previous occupations. It is applied to schemes for the psychological treatment of those who suffer from temperamental instability in conditions for which they are emotionally unsuited. We see it defined as the rehousing and resettlement of normal healthy people in new satellite towns, and of the unfit in settlements adapted to their special disabilities. Sometimes, unfortunately, it means nothing at all, but is a mere shibboleth and an evasive answer to modern problems to which the speaker has found no satisfactory answer.

The Oxford Dictionary defines rehabilitation as "the action of restoring a former status or standing." In this definition, the operative word is "former." If we accept this, it must follow that to settle a homeless worker in a new house would go only part way in the complete process; this new home must be in an area where he can follow his own trade and so maintain his full, former economic standing. To move tuberculosis sufferers to new housing estates with higher rents would be no answer to the bearing of housing in hygienic surroundings on continued resistance. Higher rents mean a lowering in the value of real wages;

a reduction in the amount of money available for food, clothing and the necessary luxuries of modern life can result in a lowering of general resistance out of all proportion to the gain in salutary air and in living space. Likewise, remedial exercise in orthopedics are comparable with the older conception of sanatorium routine in rest and graded exercise; both approximate to true rehabilitation only if they restore full previous function. They fail if they produce the maximum use of a lessened function, unless they are supplemented by a subsidy or a pension, which makes up the difference in earning power to the former pay-packet, or unless they lead to job-placement where lessened function can command that same pay-packet.

To be complete, the rehabilitation of the tuberculous person therefore implies a restoration founded on a full knowledge of the individual patient's physical, psychological, economic and social needs. Such a comprehensive scheme must be the ideal of every tuberculosis service. It is an integral part of the modern conception of treatment.

R. R. TRAIL
London, England

SCOTSON

Of late, we've gotten into a high-pitched lather about the spelling, or rather the misspelling, of the Gaelic prefix for *son of;* i.e., we were confused by the MacDonalds, the Macdonalds, the McDonalds, the M'Donalds, and even the M^{c}Donalds. When we heard that the sons of Vikings had organized counter-confusion with Anderson, AnderSon, AnderSn, Anders'n, and, so help us, Anders^{n}, we undertook some research. The variations of the Viking suffix for *male offspring of* we can't vouch for, but the Gaelic we have straight and doubly verified by one of the most scholarly, Campbell-hating Macdonalds who ever trod the valley of the Coe.

In the old days, practically no one could spell (things haven't changed so much), so to write even an insult, the Gael had to find a scribe. The scribe was a fellow Scot, of course, and not at all above economizing on ink. This economy took the form of chronic, creeping cheating which progressively reduced the D to a D, then deleted the A, and finally overwhelmed even the C. Common decency was the only thing that saved the apostrophe to stand in for the A and C. This was phase one.

Phase two came when Scots migrated to this country, Many having learned, in the meantime, to read and write. When they arrived in America they found untold numbers of McDonalds—all Irish and most of them attending a different kirk. Economy, be damned. The Scots hesitated not an instant. They spent their farthing for extra ink and reinserted the missing A's and C's in their names. And, we're told, 'tis the same story for the dir-r-r-ty Maclarens, and the bloody Macleods of Skye, and others.

The differences in spelling, then, are reduced to orthographic arrests at different evolutionary stages.

H.G.M.

DIAGNOSTIC USEFULNESS OF PERITONEOSCOPY

(Continued from Page 414)

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President's Letter

PHYSICIANS AND SOCIAL SECURITY

Many doctors in Minnesota and the nation are still asking why organized medicine is opposed to social security coverage for their profession on a compulsory basis. For a profession that has been and still is threatened with socialization, this is hard to understand. Certainly, anyone who has taken the time to read the numerous articles appearing in the J.A.M.A., our own MINNESOTA MEDICINE and special articles prepared by Dr. Frank H. Dickenson, director of the Bureau of Economic Research of the American Medical Association, should be convinced that the whole Social Security program or Old Age and Survivors Insurance program, as it is now designated, is not actuarially sound, is definitely socialism, rapidly approaching Townsendism, and is putting an enormous financial burden on our children and grandchildren for generations to come.

In the first place, OASI is not insurance. Insurance means a contract; one party agrees to provide certain returns for certain definite premiums paid in. Once the contract is made, it is binding; the rates cannot be changed; the rewards cannot be altered. But in OASI, the Act that established it definitely states in Section 1104: "The right to alter, amend or repeal any provision of this act is hereby reserved to the Congress." Through Title Two of the Social Security Act, Congress has created a system providing statutory rights to statutory benefits. Such rights are not natural, constitutional nor contractual rights.

Social Security tax rates have already risen and at present are 3 per cent of the first $4,200 of the self-employed individual's income.

Provision is now made for this to increase to 3.75 per cent from 1960 to 1964, 4.5 per cent from 1965 to 1969, 5.25 per cent from 1970 to 1974 and 6 per cent starting in 1975. On earnings of $4,200 per year, the projected raises will increase the tax from $126 per year to $252 by 1975. If the controversial H.R. 7225 pertaining to cash benefits to disabled at age fifty should pass through Congress, this percentage would rise to 3.75 per cent immediately. Already the tax base has risen from $3,600 income to $4,200 income, and there is nothing to prevent any future Congress from raising it to $4,810, $6,000 or any additional figure.

The parties pressing for OASI for physicians make much of the survivors' benefits. Let us look at survivors' benefits for a moment. A physician has a wife and two children, and a net income of $3,600 per year. He dies. His widow collects $168.75 per month until the eldest child reaches eighteen years of age; then the amount drops to $127.50 per month until the second child reaches eighteen. Then it all stops until the widow reaches sixty-five, at which time it resumes at the rate of $63.80 per month unless she remarries.

However, if this same self-employed physician were carrying a $20,000 ordinary life insurance policy, which he should be, he could add a twenty-year family income clause to his policy which in event of his death, would provide $200 per month to his family for twenty years from the date of the policy, or if he died three years after taking out such family protection, it would pay $200 monthly for seventeen years after his death. Such coverage would cost him approximately $85 additional premium on his ordinary life policy, much less than the $126 for OASI, and his wife would get this income for the stated period, whether she was gainfully em-

ployed or not and whether she remarried or not. Such insurance is a definite contract provided by extra premiums, has a definite scale of benefits and is enforceable in the courts.

The situation of the employed persons covered under OASI and those persons, now some 20 million, covered under various industrial pension plans or profit-sharing plans, is definitely different. Under the Social Security Act, both employe and employer contribute to the fund by taxation and returns to the employe in retirement benefits after reaching the age of sixty-five will amount to approximately $30 for each dollar paid into the fund.

In December, 1954, the Ways and Means Committee of the House released a report entitled "Social Security after Eighteen Years." These statements in that report are significant:

"When eligible persons aged 65-75 (now 65-72) earn too much, they must forego their benefits, as well as continue to pay OASI taxes," or:

"There is not enough in the OASI trust fund of $17 billion to pay future benefits to the present beneficiaries," or:

"Today's OASI taxpayers who become beneficiaries tomorrow must look to those working and paying OASI taxes for their benefits," or:

"Total benefits to some aged couples may aggregate several hundred times the amount they paid in OASI taxes."

This certainly does not sound like an actuarially sound insurance program.

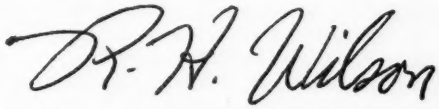
Under the many profit-sharing plans set up by companies to provide for retirement pay, the employer may set aside as much as 15 per cent of the employe's annual pay each year and not have to pay income taxes on this amount.

Under some pension plans, an employer may contribute as high as 200 per cent of the employe's annual wage, without violating the internal revenue code, that is without making the contribution currently taxable income to the employe. These contributions to pension plans by employers are tax deductible as a business expense to the employer and the employe pays no income tax on such amounts until he starts to participate in the pension benefits after retirement.

The self-employed have never had any similar opportunity to defer payment of income taxes on whatever they may be able to set aside for their retirement. The Jenkins-Keogh bills (H.R. 9 and H.R. 10, January 15, 1955), are designed to provide similar tax deferment for the self-employed up to 10 per cent of earned income in any year up to age 65.

Last month, our state office conducted a poll of all our members asking them to vote on the question, "Do you favor extension of the Old Age and Survivors Benefits coverage of the social security program to physicians? And if so, do you approve of it on a voluntary or on a compulsory basis?"

I hope the information provided here will help you decide whether you want to participate in such a program or whether you want to provide your own retirement fund, aided in so doing by adoption of some plan of deferred income tax such as that incorporated in the Jenkins-Keogh bills.



President, Minnesota State Medical Association

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

FORD FOUNDATION GIVES $10 MILLION TO FUNDS FOR MEDICAL SCHOOLS

A $10 million program of grants to the National Fund for Medical Education was announced in mid-April by H. Rowan Gaither, Jr., president of the Ford Foundation. The National Fund for Medical Education is the organization which distributes to the medical schools the funds raised by the American Medical Education Foundation along with the contributions made by industry and the general public.

Gaither said the Ford Foundation grants will be made on a matching basis over a five to ten-year period with a maximum grant in any one year limited to $2 million.

In 1955 the National Fund received $2,147,000 in unearmarked funds for distribution to the nation's medical schools. Of this amount, an unearmarked $422,812 came from the medical profession through the American Medical Education Foundation. Under the Ford Foundation formula, if 1956 receipts are equally large, there would be a Ford grant totaling 70 per cent of this amount or $1,503,486. All contributions in excess of the 1955 total would be matched dollar-for-dollar, subject to the annual maximum of $2 million.

After 1956, each year's grant would match the preceding year's receipts on a diminishing scale but all receipts over the preceding year's total, would be matched dollar-for-dollar up to the $2 million ceiling.

The announcement of these matching grants is heartening to physicians who have contributed to the medical schools through the A.M.E.F. It will mean that for every unearmarked dollar contributed to A.M.E.F., another 70 cents to a dollar will be given by the Ford Foundation, thereby stimulating the additional support the nation's medical schools have so desperately needed.

WHY HOSPITAL COSTS ARE HIGH

After a careful analysis of the period from 1946 to 1954, Ray E. Brown, president of the American Hospital Association, recently explained why hospital costs are high. He also predicted that "hospital costs would continue to increase at about five per cent annually for many years."

His article, "The Nature of Hospital Costs" appears in the April 1, 1956, issue of *Hospitals,* the journal of the American Hospital Association.

"Whatever answers there are to the problem of hospital costs must be found in the area of personnel budgets," he said.

Contrasting the hospital as a personal service institution with general industry, Mr. Brown wrote:

"The nature of the hospital's work provides little opportunity for . . . productivity gains. Nonetheless, hospital salary levels are affected by general salary levels. Under such circumstances every round of salary increases constitutes a direct increase in hospital costs. Over the years ahead, as the general labor force continues to receive its rightful share of the proceeds from increased productivity, hospitals will be compelled to give equal increases in salary without comparable benefits from increased productivity."

NEW AMERICAN MEDICAL DIRECTORY COMPLETED

After twenty months of work, the 19th edition of the American Medical Directory has been completed. The price is $30 per copy including postage within the United States. Orders may be placed by writing Philip E. Mohr, Directory editor, American Medical Association, 535 North Dearborn Street, Chicago 10, Illinois.

The new directory shows a 16,784 gain in the total number of physicians since 1950. The Pacific states show the largest increase with a gain of 23 per cent over the 1950 figures. States showing a substantial increase in the number of physicians include California, Florida, Texas, Ohio, Michigan and New York. Slight losses in number occurred in Arkansas, Illinois, Iowa, Missouri, Vermont and West Virginia.

The Directory also shows that 30 per cent of the physicians in the United States are in general practice; 10 per cent give special attention to a specialty but do not limit their practice to it; 31 per cent limit their practice to a specialty; 11 per cent are serving internships or residencies; 6 per cent are in other full-time hospital services, 5 per cent are retired or not in practice; 4 per cent are not in private practice and 3 per cent are temporarily in military service or serving in various government agencies.

JUDD SPEAKS AT SAMA MEETING

Minnesota Congressman Walter Judd spoke at the sixth annual Student American Medical Association convention at the Sherman Hotel, Chicago, May 5. His subject was "The Doctor's Place in Public Affairs."

The AMA honored the 1956-57 officers and councilors of the SAMA at a reception Sunday, May 6.

SENATE COMMITTEE APPROVES TWO HEALTH BILLS

The Senate Public Welfare Committee has unanimously approved two health bills. One would authorize the Public Health Service to undertake the continuing collection of data on the prevalence and incidence of the major categories of sickness and disability.

The second bill would authorize $5 million for the construction of a building to house the National Institute of Dental Research.

TWO MEASURES MAY AFFECT AGED

President Eisenhower has established a Federal Council on Aging. This Council will consist of representatives of 12 Federal agencies which have a special concern with aging. It will have a general co-ordinating function in an effort to achieve a more effective government-wide approach to the needs of older persons.

A bill has been reintroduced by Representative John D. Dingell (D., Mich.,) affecting hospitalization of the aged. Its provisions, in brief, call for a maximum of 60 days free hospitalization a year if ordered by a physician and for any social security beneficiary or dependent. Excluded would be surgery or medical care (except that generally furnished by a hospital), tuberculosis, mental conditions or domiciliary or nursing type care. Included would be nursing and lab, drug and operating room costs. The Secretary of HEW would make regulations with advice from the Federal Hospital Council. Operating as agents of the U. S., states would make arrangements with hospitals. Payment would come out of the OASI Trust Fund without additional tax.

NEW VERSION OF ALASKAN MENTAL HEALTH BILL PROPOSED

Senator Goldwater (R., Ariz.,) has drawn up a bill that would amend the House-passed Alaskan Mental Health Bill. It would amend the House measure to give the Territory of Alaska the responsibility of working out commitment procedures. The commitment section of the House bill produced considerable opposition from some groups. The AMA favored the House-passed bill but as yet has not passed on the Goldwater proposal.

OFFICE OF CIVIL AVIATION MEDICINE PROPOSED

The Chairman of the House Interstate and Foreign Commerce Committee has proposed that the Administrator of Civil Aeronautics be given authority over medical aspects of civil aviation including pilot examinations and the establishment of minimum human requirements in aircraft design. These are currently the responsibility of the Civil Aeronautics Board. An Office of Civil Aviation Medicine would be set up within CAA as well as a research laboratory and a medical advisory committee.

U. S. WARNS PUBLIC AGAINST HOXSEY CANCER TREATMENT

For the first time in recent years, the Food and Drug Administration issued a public warning . . . to "sufferers from cancer, their families, physicians and all concerned with the care of cancer patients" to avoid the Hoxsey treatment developed by Harry M. Hoxsey, of Texas.

The public warning says in part:

"The Food and Drug Administration has conducted a thorough and long-continuing investigation of Hoxsey's treatment. His claimed cures have been extensively studied and the FDA has not found a single verified cure of internal cancer effected by the Hoxsey treatment."

FDA Commissioner George P. Larrick points out that Harry Hoxsey and "all persons in active concert with him" in the treatments were enjoined October 26, 1953, from shipping their "worthless cancer medicines" in interstate commerce with labeling representing or implying that the products are effective in the treatment of any type of internal cancer.

While the U. S. plans prosecution of violations of the injunctions, Mr. Larrick says the warning is issued "for the immediate protection of cancer victims. . . ."

MILITARY MEDICAL LEGISLATION BILLS GO THROUGH HOUSE

Two bills concerning military medical legislation went through the House without change recently after detailed hearings and study by a subcommittee. One is designed to make military medical careers more attractive by allowing credit for time spent in medical school and internship and by setting up a series of three $50 per month raises after three, six and ten years' service. These would be in addition to the present $100 per month special pay for medical officers. Public Health Service medical officers would benefit as well as those in the Army, Navy and Air Force.

The other bill well on its way to becoming a law allows dependents of servicemen to receive private hospital and medical care with the government paying the cost of the insurance or health plan coverage and the dependent the first $25 of the hospital bill. The Secretary of Defense could limit or deny such private care in areas where he determines that military medical facilities are adequate to handle the service families.

FEE COMPLAINTS NOT NECESSARILY BASED ON FEES

The Mediation Committee of the Wayne County (Mich.) Medical Society found that fee complaints are less likely to arise from the size of the fee than from the doctor's failure to explain his services. This group completed a study of the reasons behind 56 recent protests to the committee.

In only three of those cases, it reports, did patients refuse to pay because they considered the fees themselves unreasonable.

Sixty per cent of the patients with grievances balked at paying because, in their opinion, the physician in the case had rendered them no real service. In another 25 per cent of the cases, fee complaints appear to have been caused by "insurance problems and improper billing procedures."

The final 15 per cent of the fee complaints reportedly stemmed from patients' doubts about the quality of service they had received.

Dr. Luther Leader, chairman of the committee, concluded that "half the cases were settled by explanation only. And how much more satisfactory for all concerned if these explanations had come from the physician rather than the committee. If each doctor had spent more time with each patient, there would have been little left for the mediation committee to do."

AMA LAW DEPARTMENT SPONSORS MEETING

Attorneys representing medical societies gathered in Chicago for a two-day meeting in April. This was the first formal full-scale attempt to bring together attorneys who are called upon to advise medical society clients on their many legal problems. Panel discussions were conducted on antitrust laws and related problems, the corporate practice of medicine, grievance committees and disciplinary problems, professional liability, interprofessional codes of conduct and legislative and lobbying activities.

MEDICAL ASSISTANCE BILLS INTRODUCED

Administration-sponsored amendments to the Social Security Act which would provide separate matching by the states of public assistance payments for medical care were taken up by the House Ways and Means Committee in April. Bills are sponsored by Representative Jere Cooper, (D., Tenn.,) and Representative Dan Reed, (R., N.Y.,) in the House and by Senator Martin (R., Pa.,) in the Senate. Under these bills the federal contribution to a state for medical care would be $3 a month per adult recipient and $1.50 for a child, with the state required to match dollar-for-dollar. Under the present setup public assistance payments going to the states are not specifically earmarked for medical care.

COMMISSIONS FOR OSTEOPATHS STILL IN DOUBT

A bill to give osteopaths military commissions has bogged down temporarily in the Senate Armed Services Committee. The Defense Department's medical chief, Dr. Frank Berry, backs the idea, but his Service surgeons-general all oppose it.

GOVERNMENT INVESTIGATES ABUSES IN VA OUTPATIENT PROGRAM

The medical profession came out with a nearly clean bill of health in the government's recent survey on abuses in the outpatient program of the Veterans Administration. The survey disclosed that a number of physicians had made mistakes in charging for the treatment of veterans but only a few deliberately cheated. Representative Olin Teague, (D., Tex.,) chairman of the House Veterans Committee which demanded the survey, said the abuses ranged from "minor administrative failures to serious false claims" and referred the cases to the Justice Department for prosecution.

"Certainly these abuses do not provide a basis for the generalization that there is dishonesty in the medical profession," he said. "They do, however, show that in any large program involving many thousands of individuals a certain amount of carelessness, misunderstanding and, in a few cases, outright dishonesty will creep in."

Meanwhile the Veterans Administration says it has not found many abuses in its disability compensation and pension programs. In an investigation of more than 400,000 cases, it has found nearly 96 per cent of the claims entirely accurate.

THE MIGHTY MUSHROOM

In Central Europe and in Pennsylvania, folklore credits the eating of certain species of mushrooms with protective effect against development of human cancers. Extracts from ten of eighty species examined do indeed demonstrate some inhibitory effect upon transplanted cancers in mice, report Dr. E. H. Lucas and Joseph Stevens, Michigan State University. The extracts apparently contain some cancer-retarding factor, according to the studies done in collaboration with the Sloan-Kettering Institute, New York. The factor is apparently heat resistant. None of the extracts has yet been purified sufficiently for human experiments, and any role in providing clues toward cancer control is yet unknown.—Medigrams: *GP* 12:240 B (Oct.) 1955.

In Memoriam

WILLIAM H. CONDIT

Dr. William H. Condit, treasurer of the Minnesota State Medical Association since 1932, died April 21, 1956. He was eighty-four years old.

A long-time specialist in obstetrics, gynecology and surgery, Dr. Condit had practiced medicine in Minneapolis since 1899.

He graduated from the University of Minnesota Medical School and interned at St. Barnabas Hospital, Minneapolis. He became a fellow of the American College of Surgeons in 1922 and joined the Nicollet Clinic, Minneapolis, in 1924. He was also a teaching assistant at the University of Minnesota Medical School.

Dr. Condit was a former vice president of the Minnesota State Medical Association and in 1952 the Association awarded him its highest honor, the distinguished service award.

He had been a staff doctor at Northwestern, Doctors Memorial, and University Hospitals.

His memberships included the Academy of Medicine, the American Medical Association, the Southern Minnesota Medical Association and the Hennepin County Medical Society.

Survivors include a sister-in-law, Mrs. Mayme Dann, Minneapolis. Dr. Condit's wife died many years ago. Interment was in Albany, New York.

EVERETT C. HARTLEY

Dr. Everett C. Hartley, St. Paul obstetrician and gynecologist, died May 2, 1956. He was sixty-five years old.

A native of Carver, Minnesota, Dr. Hartley graduated from Beloit College, Beloit, Wisconsin, and received his medical education at the University of Minnesota. He did postgraduate work in Berlin and during World War I he served with the 26th Medical battalion in France. After the Armistice, Dr. Hartley was attached to the Red Cross in Poland for two years.

An associate professor at the University of Minnesota medical school, he was a member of the Ramsey County Medical Society, the Minnesota State Medical Association and the American Medical Association. He also belonged to the Central States Association of Obstetrics and Gynecology, the Minnesota Association of Obstetrics and Gynecology and the Travel Club.

Dr. Hartley was a consultant in maternal and child health for the division of child welfare, Minnesota Department of Health.

Survivors include his wife, Zita; a son, Everett Charles, Jr., two daughters, Jane and Mrs. Harold L. Korn, New York City, and a brother, Scott, Carver, Minnesota.

PAUL A. HIGBEE

Dr. Paul A. Higbee, who practiced medicine in Minneapolis nearly fifty years, died March 29, 1956. He was eighty-two years old.

Born in Red Wing, he attended the University of Minnesota where he was a member of Delta Kappa Epsilon fraternity. He was graduated from Hahnemann Medical College, Philadelphia, and served as a captain in the army medical corps during World War I.

He was a member of Khurum Masonic Lodge and of Scottish Rite bodies of Minneapolis and was a 32nd degree Mason.

Dr. Higbee was a member of the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Surviving are his wife, Louise, and two sisters, Mrs. Earle G. Nunnally, Cincinnati, Ohio, and Mrs. Reginald J. Healy, Minneapolis.

RUFUS O. JOHNSTON

Word has just been received of the death last December of Dr. Rufus O. Johnston, former physician for Independent School District Number Twenty-Seven, Hibbing, Minnesota.

Dr. Johnston died in Florida. He was fifty-four years old.

A graduate of the University of Minnesota Medical School, he practiced in Harrington Park, New Jersey, for a number of years and served as a medical officer in the U. S. Army during World War II.

He was a member of the St. Louis County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Dr. Johnston is survived by his wife and two daughters.

HENRY W. NOTH

Dr. Henry W. Noth, a retired staff member of St. Barnabas Hospital, Minneapolis, died March 27, 1956. He was seventy-eight years old.

Born in Maple Grove, Dr. Noth practiced medicine in Minnesota for more than fifty years. In 1953, he was inducted into the fifty-year club of the Minnesota State Medical Association.

He was also a member of the Hennepin County Medical Society, the American Medical Association, Hennepin Masonic Lodge Number 4, Scottish Rite Temple and Izaak Walton League.

Dr. Noth graduated from the University of Minnesota Medical School in 1903.

Surviving are a son, Dr. Paul H. Noth, Los Alamos, New Mexico; a daughter, Mrs. Katherine Chambers, and a sister, Mrs. Albert McKenzie, both of Minneapolis.

HOMER M. McINTIRE

Dr. Homer M. McIntire, pioneer Waseca, Minnesota, physician, died April 18, 1956. He was seventy-one years old.

Born in Winchester, Ohio, he attended Park Academy

(Continued on Page A-34)

Meetings and Announcements

MEDICAL MEETINGS

State

Minnesota Society of Obstetrics and Gynecology, fall meeting, Rochester, Minnesota, September 22. Dr. Edward A. Banner, secretary, Rochester, Minnesota.

National

American Association of Blood Banks, ninth annual meeting, Somerset Hotel, Boston, Massachusetts, September 3-5. Secretary Marjorie Saunders, 725 Doctors Building, 3707 Gaston Ave., Dallas, Texas.

American Heart Association, 29th annual scientific session, Cincinnati, Ohio, October 26-29. Write American Heart Association, 44 East 23rd Street, New York 10, New York.

American Society of X-Ray Technicians, annual convention, Kentucky Hotel, Louisville, Kentucky, June 17-21.

Minnesota-Dakota-Manitoba Orthopedic Society, annual meeting, Red Wing, Minnesota, September 7 and 8. Dr. Hollis Ahlrin, secretary, Rapid City, South Dakota.

National Society for Crippled Children and Adults, annual convention, Hotel Statler, Washington, D. C., October 28-31.

Postgraduate Medical School of New York University—Bellevue Medical Center, eight-week course in occupational medicine, September 10-November 2. Write Dean, N.Y.U. Postgraduate Medical School, New York 16, New York.

Symposium for General Practitioners on Tuberculosis and Other Chronic Pulmonary Diseases, fifth annual meeting, Saranac Lake, New York, July 7-13. Dr. Edward N. Packard, general chairman, Symposium for General Practitioners, P.O. Box 262, Saranac Lake, New York.

University of Illinois Department of Otolaryngology, Annual Assembly in Otolaryngology, October 1-7. Write Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Illinois.

West Virginia State Medical Association, 89th annual meeting, The Greenbrier, White Sulphur Springs, West Virginia, August 23-25.

International

Congress of International Society of Hematology, Hotel Somerset, Boston, Massachusetts, August 27-September 1. Dr. W. C. Moloney, secretary, 39 Bay State Road, Boston, Massachusetts.

European Congress of Cardiology, Stockholm, Sweden, September 10-14. Dr. Karl E. Grewin, secretary, Sodersjukhuset, Stockholm, Sweden.

Inter-American Congress of Cardiology, Havana, Cuba, November 4-10. Dr. Ramon Aixala, Apartado 2108, Havana, Cuba.

International Congress of Clinical Chemistry, Hotel New Yorker, New York, September 9-14. Secretary, John G. Reinhold, 711 Maloney Building, University of Pennsylvania, Philadelphia 4, Pennsylvania.

International Congress of Gastroenterology, London, England, July 18-21. Herman Taylor, secretary, London Hospital, White Chapel, London E. 1, England.

International Congress of Internal Medicine, Madrid, Spain, September 19-23. Dr. J. C. DeOya, secretary, No. 90, Madrid, Spain.

International Congress of International College of Surgeons, Palmer House, Chicago, Illinois, September 9-13. Dr. Max Thorek, secretary general, 1516 Lake Shore Drive, Chicago, Illinois.

International Congress of Radiology, Mexico, D. F., Mexico, July 22-28. Dr. Jose Noriega, secretary, Tepic 126 (2 e piso), Mexico, D. F. 7, Mexico.

International Congress on Diseases of the Chest, Cologne, Germany, August 19-23. Murray Kornfeld, executive director, 112 East Chestnut St., Chicago 11, Illinois.

Pan American Congress on Cancer Cytology, Miami, Florida, January 8-12, 1957. Dr. J. Ernest Ayre, chairman, 1155 N. W. 14th St., Miami, Florida.

World Medical Association, Havana, Cuba, October 9-15. Dr. Louis H. Bauer, secretary general, 345 East 46th St., New York 17, New York.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

October 1 is the deadline date for receipt of applications for the 1957 Part I examination for the American Board of Obstetrics and Gynecology. Further information can be obtained from Dr. Robert L. Faulkner, Secretary, American Board of Obstetrics and Gynecology, 2105 Adelbert Road, Cleveland 6, Ohio.

AMERICAN COLLEGE OF PHYSICIANS

The American College of Physicians held its 37th annual session at Los Angeles, California, April 16-20. Elected to membership in the group were Dr. Norman A. Christensen, Dr. John E. Estes, Dr. Donald R. Nichols and Dr. Maurice H. Stauffer, all of Rochester; Dr. Benjamin F. Fuller and Dr. Herbert W. Johnson, both of St. Paul; Dr. William B. Martin, Duluth; Dr. Neil D. Nickerson, Fairmont, and Dr. Harold E. Miller, Minneapolis.

AMERICAN HEART ASSOCIATION PROCEEDINGS PUBLISHED

Proceedings of the American Heart Association's 1955 annual meeting of the Council for High Blood Pressure Research, held in Cleveland last November, have been published in book form. Containing eight papers and verbatim transcripts of discussions, the proceedings offer a roundup of recent knowledge in the field of hypertension, with emphasis on neurovascular aspects. Further information is available from the American Heart Association, 44 East 23rd Street, New York 10, New York.

INTERNATIONAL COLLEGE OF SURGEONS SCHOLARSHIPS

The awarding of three $3,000 scholarships for postgraduate study in surgery has been announced by the United States and Canadian sections of the International College of Surgeons. The funds were made available by the Woman's Auxiliary to the two sections. The auxiliary also has made funds available to pay the expenses, not exceeding $300, of two residents or fellows in training as surgeons to enable them to attend the 21st annual Congress of the United States and Canadian sections in Chicago, September 9-13, and to present suitable clinical or scientific papers before the assembly. Information may be obtained from the International College of Surgeons, 1516 Lake Shore Drive, Chicago 10, Illinois.

INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST

The fourth International Congress on Diseases of the Chest of the American College of Chest Physicians will be held in Cologne, Germany, August 19-23. Eighty-six countries will send representatives to the meeting. The congress will place special emphasis on surgery for coronary diseases. For detailed information, write Fourth International Congress of the American College of Chest Physicians, Köln-Deutz, Germany, Messeplatz.

MINNESOTA SOCIETY OF NEUROLOGY AND PSYCHIATRY

The Minnesota Society of Neurology and Psychiatry held a morning meeting at Rochester on May 19. A program of surgical clinics at St. Mary's Hospital was followed by a scientific session, at which papers were presented by Dr. Alfred Uihlein, Dr. Henry W. Dodge, Jr., Dr. J. G. Rushton, Miss Josephine Simonson, Dr. Howard P. Rome, Dr. D. W. Mulder and Dr. David D. Daly. At a luncheon in the Kahler Hotel the principal speaker was Dr. C. H. Hodgson, who spoke on "Africa, the Changing Continent."

NORTHERN MINNESOTA MEDICAL ASSOCIATION MEETING

The annual meeting of the Northern Minnesota Medical Association will be held at Alexandria on September 7 and 8. The meeting will begin on Friday at 10:00 a.m. and continue until Saturday noon. Scientific sessions will be held in the auditorium of the Alexandria Clinic. A detailed program will be published later.

MINNESOTA MATERNAL MORTALITY STUDY

(Continued from Page 386)

adequacies of death certificate reporting are again apparent in this study.

7. Autopsies were obtained in 49.2 per cent of the deaths. This shows considerable improvement over previous studies and reflects more effort and interest in the study of these cases by the local physicians.

8. The primary causes of maternal deaths in Minnesota in the 1953 study are tabulated. Toxemia was the leading cause as it was in 1950. Deaths due to hemorrhage headed the list in 1951 and 1952. Three of the deaths due to toxemia were considered to be preventable.

9. The hemorrhage deaths are discussed briefly. All of these were considered preventable.

10. The dangers of carelessness in transfusion technique are emphasized by the deaths due to transfusion accidents. These equaled the number due to hemorrhage. These two causes combined accounted for as many deaths as did toxemia.

References

1. Maternal Mortality Committee, Minnesota State Medical Association: Maternal Mortality Study, July 1, 1941, through June 30, 1942. Minnesota Med., 27:475-481; 557-562, 1944.
2. Maternal Mortality Committee of the Committee on Maternal Health of the Minnesota State Medical Association: Minnesota Maternal Mortality Study. Minnesota Med., 36:609-622 (June) 1953.
3. Maternal Mortality Committee of the Committee on Maternal Health of the Minnesota State Medical Association; Minnesota Maternal Mortality Study. Minnesota Med., 37:131-135 (Feb.) 1954.
4. McKelvey, J. L., and Freeman, D. M.: Critical Evaluation of the Minnesota Maternal Mortality Study. Am. J. Obst. & Gynec., 68:29-37 (July) 1954.
5. Maternal Mortality Committee of the Committee on Maternal Health of the Minnesota State Medical Association: Minnesota Maternal Mortality Study. Minnesota Med., 38:9-15 (Jan.) 1955.

General Interest

Dr. Fred W. Wittich, Minneapolis, presented a paper at the thirteenth annual meeting of the American Geriatrics Society at the Palmer House in Chicago on May 3 and 4. Dr. Wittich recently retired from active duty with the American College of Allergists and now is devoting his time entirely to his private practice and editing the *Quarterly Review of Allergy and Applied Immunology.* At the twelfth annual meeting of the American College of Allergy, held recently in New York, Dr. Wittich was made president emeritus of the organization.

* * *

Dr. Reno W. Backus, staff member of Nopeming Sanatorium, Nopeming, since 1943, has been named superintendent and medical director of the sanatorium. He succeeds **Dr. G. A. Hedberg,** who died in March. A graduate of Rush Medical College in 1925, Dr. Backus began practice in 1928 in Peking, China, where he served as superintendent of the Hopkins Memorial Hospital until 1941.

* * *

New chairman of the St. Paul Area Health Council is **Dr. Frederick M. Owens, Jr.,** St. Paul.

* * *

Dr. H. Herman Young, Rochester, chairman of the orthopedic surgery sections of the Mayo Clinic, was toastmaster at the Minnesota Safety Council's annual award presentation dinner in Minneapolis on May 1. Dr. Young, a member of the executive committee of the council, has been active in the control and prevention of farm and industrial accidents.

* * *

Dr. Lewis Hanson, Frost, was elected chief-of-staff of the Blue Earth Community Hospital at the annual election meeting of the hospital staff recently. He replaces **Dr. H. H. Russ,** Blue Earth. Named as secretary-treasurer was **Dr. James A. Anderson,** who recently joined the staff of the Blue Earth Medical Center.

* * *

Dr. William B. Tucker, former chief of the tuberculosis service at Minneapolis Veterans Hospital, will become director of the VA tuberculosis service in medicine and surgery in Washington, D. C., on September 1. For the past two years since leaving Minneapolis Dr. Tucker has been chief of the medical service at the veterans hospital at Durham, North Carolina.

* * *

At the 53rd semi-annual Postgraduate Extension Course at Marshall on April 17, **Dr. Merrill D. Chesler,** Minneapolis, spoke on "The Treatment of Burns in Children; the Treatment of Birthmarks and Related Congenital Abnormalities." The extension course was sponsored by the University of Minnesota, the Lyon-Lincoln County Medical Society and the Minnesota State Medical Association.

Dr. Chesler also was the principal speaker at a meeting of the Kandiyohi-Swift-Meeker County Medical Society in Willmar on April 19, where his topic was "Practical Applications of Skin Grafts and Flaps."

* * *

Dr. James T. Priestley, Rochester, recently received an honorary doctor of laws degree from the University of Alberta.

* * *

Dr. John S. Milton has been made a member of the board of trustees of Swedish Hospital, Minneapolis. **Dr. A. E. Johnson** has been re-elected treasurer of the board.

* * *

Dr. Jan H. Tillisch, Rochester, was elected president of the Aero Medical Association at the annual meeting of the organization in Chicago in mid-April. Dr. Tillisch, who was in the Air Force during World War II, is a member of the International Air Transport Medical Committee and is associated with the American Board of Aviation Medicine.

* * *

Principal speaker at tuberculosis accreditation ceremonies at Lowell School in Brainerd on April 17 was **Dr. M. M. Williams,** superintendent and medical director of the state sanatorium at Ah-Gwah-Ching. The program consisted of accreditation of Crow Wing County in recognition of its progress in reducing rates of infection and deaths from tuberculosis. The accreditation was sponsored by the Minnesota State Medical Association, the Minnesota Department of Health and the Christmas Seal organization.

* * *

Dr. Wesley W. Spink, professor of medicine at the University of Minnesota, presented the annual address at a meeting of the Minneapolis Academy of Science in Minneapolis on May 4 and 5. The title of his talk was "Undulant Fever, an Example of the Interdependence of Research."

* * *

Dr. J. G. Love, Rochester, member of the neurological surgery staff of the Mayo Clinic, presented a paper at the centennial celebration and scientific congress of the Hawaii Medical Association in Hawaii April 22 to 29. The title of the paper, which was written by Dr. Love, **Dr. H. W. Dodge, Jr.,** and **Dr. C. M. Gottlieb,** was "Benign Tumors of the Foramen Magnum."

* * *

Special award keys honoring former chairmen of the Ramsey County chapter of the National Foundation for Infantile Paralysis were presented at a luncheon in St. Paul on April 12. Among those presented keys, plus fifteen-year pins for service to the national foundation, were **Dr. Walter R. Ramsey, Dr. C. C. Chatterton** and **Dr. James Dunn,** all of St. Paul.

* * *

At a meeting of the six county Southwestern Minnesota Medical Society at Worthington on April 23, the

(Continued on Page A-30)

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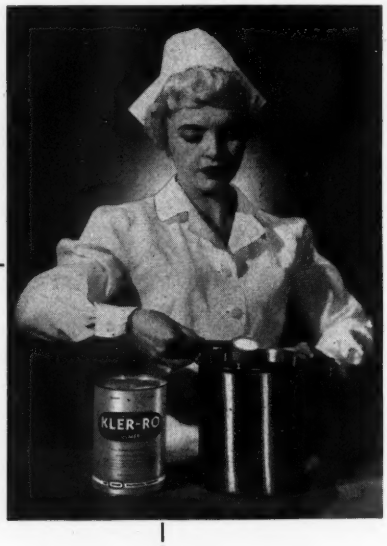

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(Continued from Page 432)

principal speakers were **Dr. E. Harvey O'Phelan** and Dr. **W. R. Schmidt,** both of Minneapolis. Dr. O'Phelan spoke on "Fractures in Children," while Dr. Schmidt discussed "Chest Injuries."

* * *

Dr. John R. McDonald, Rochester, head of a section of surgical pathology in the Mayo Clinic, has been awarded the 1956 medal for Minnesota by the American Cancer Society. The award was for Dr. McDonald's research work in the early detection of cancer cells in various body fluids. He has been at Rochester since 1933, when he first became a fellow in pathology at the Mayo Foundation.

* * *

Speakers at the fourth annual Upper Midwest Business and Industrial Forum on Problem Drinking, held in Minneapolis on May 9, included **Dr. Nelson J. Bradley,** superintendent of Willmar State Hospital, Willmar, and **Dr. Robert L. Faucett,** Rochester.

* * *

Dr. Howard P. Rome, Rochester, head of psychiatry section at the Mayo Clinic and professor of psychiatry in the Mayo Clinic, was elected for a three-year term on the governing council of the American Psychiatric Association at the annual meeting of the organization in Chicago on April 30.

* * *

Dr. Frank M. Manson, retired Worthington physician, celebrated his eighty-fifth birthday on April 10. Dr. Manson began practice in Worthington in 1899 and started its first hospital in 1906. In 1939, due to severe radiation burns on his hands from long exposure to x-rays, Dr. Manson retired from active practice.

* * *

The Friedenwald Medal of the American Gastroenterological Association was awarded to **Dr. Donald C. Balfour,** Rochester, director emeritus of the Mayo Foundation, at the organization's annual meeting in Atlantic City, New Jersey, on April 28. The medal was created to honor outstanding contributions to knowledge in the field of gastroenterology.

* * *

Dr. Kathleen Jordan, Granite Falls, discussed tuberculin testing techniques on the "Doctor's Round Table" television show on May 6 over WCCO-TV, Minneapolis.

* * *

Dr. Gordon R. Kamman, St. Paul, and attorney **Charles Murnane** spoke on "Medical-Legal Relationships" at a joint meeting of the Lyon-Lincoln County Medical Society and the Lyon-Lincoln Bar Association at Marshall on May 7.

* * *

Dr. Waltman Walters, Rochester, head of a section of surgery at the Mayo Clinic, was formally invested with the rank of rear admiral in the United States Naval Reserve at ceremonies in Rochester on April 18.